

COPPER HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by Ronald Eisler

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Schroeder et al. 1966; 7, Yoshimura et al. 1995; 8, Gordon et al. 1990; 9, Bires and Vrzgula 1990; 10, Howell et al. 1991; 11, Lewis et al. 1967; 12, Gopinath and Howell 1975; 13, Buckley and Tait 1981; 14, Gamcik et al. 1990; 15, Aaseth and Norseth 1986; 16, Doherty et al. 1969; 17, Saari et al. 1994; 18, Panemangelore 1993; 19, Petering et al. 1977; 20, Moffitt and Murphy 1973; 21, Lal and Sourkes 1971; 22, Jehan and Motlag 1994; 23, Ng and Liu 1990; 24, Dodds-Smith et al. 1992a; 25, Dodds-Smith et al. 1992b; 26, Higgins 1981; 27, Romeu-Moreno et al. 1994.

Terrestrial Plants and Invertebrates

Copper is toxic to sensitive plants when plant nutrient solutions contain greater than 40-200 µg Cu/L, when leaves have greater than 10 to 12 mg Cu/kg DW, and when extractable copper in soils is greater than 60 mg/kg DW soil (Table 4). Excess copper inhibits root elongation and branching and reduces the ability of the plant to explore the soil for water and nutrients (Arduini et al. 1995). Root damage occurs in pine seedlings (Pinus spp.) after exposure for 10 days to nutrient solutions that contain 40 µg Cu/L. A lower concentration of 4 µg Cu/L has no adverse effects on root growth and morphology, while a higher concentration of 400 µg Cu/L completely inhibits root growth within 3 days (Arduini et al. 1995). Poultry litter is a useful agricultural byproduct with high nitrogen and phosphorus content and is frequently added to agricultural soils. Poultry litter from northern Georgia containing 1,196 mg Cu/kg DW and applied at a final rate of 5-15 mg Cu/kg soil to fields of Sudex (Sorghum bicolor x S. sudanense) did not affect copper levels of treated Sudex or produce any evidence of toxicity (van der Watt et al. 1994). But most terrestrial vegetation in the United States, Sweden, Wales, and other locales is usually adversely affected by emissions from copper mines, brass foundries, and copper smelters (Hutchinson 1979). Damage to vegetation persists for at least 50 years after closure of a copper smelter because of copper, arsenic, and lead in the soil. Particularly sensitive to copper in the soils are white pine (Pinus strobus) and red maple (Acer rubrum); less sensitive are Douglas fir (Pseudotsuga menziesii) and lodgepole pine (Pinus contorta; Hutchinson 1979).

Earthworms (*Eisenia fetida*) held in soils containing 53 mg Cu/kg DW show a 50% reduction in cocoon production in 56 days; 32 mg Cu/kg soil had no effect on cocoon production (Spurgeon et al. 1994). The LC50 (56 days) value for earthworms is 555 mg Cu/kg DW soil; no deaths occur at 210 mg/kg soil during this period. Copper is more toxic to *Eisenia fetida* than are salts of cadmium, zinc, or lead (Spurgeon et al. 1994). Copper adversely affects the earthworm *Lumbricus rubellus* (Ma 1984). Concentrations of 150 mg Cu/kg surface soil from an accidental spill of copper sulfate in grasslands reduced earthworm populations by about 50%; surface soil concentrations of 260 mg Cu/kg kill almost 100% of the *Lumbricus*. Copper is most toxic to *Lumbricus* at low soil pH (4.8-7.1) and at low temperatures (Ma 1984).

Tests show that the presence of soil reduces the toxicity of copper to the soil-dwelling nematode *Caenorhabditis elegans*; copper toxicity to nematodes increases with increasing densities of bacteria and increasing concentrations of sodium chloride or potassium chloride (Donkin and Dusenbery 1993). Terrestrial isopods efficiently assimilate and store copper as detoxified granules in the hepatopancreas; this activity is in contrast to many species of marine crustaceans that are unable to assimilate, detoxify, or otherwise regulate copper (Weeks and Rainbow 1993).

Aquatic Organisms

Plants

Photosynthesis and growth in sensitive species of freshwater algae are inhibited by copper concentrations of 1-6 μ g/L (NAS 1977; Table 5). For sensitive species of estuarine phytoplankton, copper is lethal at 50 μ g/L and most toxic under conditions of decreasing salinity, pH, and concentrations of chelators (Erickson et al. 1970). Sensitivity to copper varies widely among species of estuarine algae (Erickson et al. 1970; Table 5); some species, for example, grow normally at concentrations as high as 10 mg Cu/L during exposure for 9 days (Piccinni and Copellotti 1982). In mesocosm studies, 50 μ g Cu/L caused a reduction of about 80% in total zooplankton and total algal biovolumes; the algal assemblage that persisted was dominated by diatoms (Havens 1994). Copper-resistant strains of *Euglena gracilis* challenged with high sublethal concentrations of copper for 5 days had an altered cysteine metabolism (Coppellotti 1989).

Some species of aquatic plants absorb or adsorb dissolved copper at extremely high rates (Table 5). Bioconcentration factors for copper and freshwater alga (*Chlorella* sp.) range from 203-2,000 after exposure for 14 to 30 h (USEPA 1980). Seagrass (*Heterozostera tasmanica*) in seawater containing 42 µg Cu/L for several

| Home | Applications Processes | Systems | Products Library | Languages |
|---------------------------------------|--|---|------------------------------|--------------|
| out Lenntech | Home > Periodic table > Elements > Arsenic | | | Elements |
| rnkey plants | Arsenic - As | | | Actinium |
| ot plants | | | | Silver |
| ntainerized plants | Chemical properties of arsenic - Healt | h effects of arsenic - Environm | ental effects of arsenic | Aluminum |
| rvices | Atomic number | 33 | | Americium |
| reer at Lenntech | Atomic mass | 74.9216 g.mol ⁻¹ | | Argon |
| ternational Internships | | | | Arsenic |
| riodic table | Electronegativity according to Pauling | 2.0 | N N | Astatine |
| Iculators | Density | 5.7 g.cm ⁻³ at 14°C | | Gold |
| sitors information | Melting point | 814 °C (36 atm) | | Boron |
| ır partners | Boiling point | 615 °C (sublimation) | | Barium |
| ntact us | Vanderwaals radius | 0.139 nm | | Beryllium |
| ntact as | Validel Waais Taulus | | | Bohrium |
| | Ionic radius | 0.222 nm (-2) 0,047 nm (+5) 0,058 (+3) | | Bismuth |
| Request a Ouote ? | Isotopes | 8 | | Berkelium |
| 4 00.0 | | · · · · · · · · · · · · · · · · · · · | | Bromine |
| | Electronic shell | [Ar] 3d ¹⁰ 4s ² 4p ³ | | Carbon |
| SHARE BES. | Energy of first ionisation | 947 kJ.mol ⁻¹ | | Calcium |
| | Energy of second ionisation | 1798 kJ.mol ⁻¹ | · · | Cadmium |
| | | | | Cerium |
| tourteet BV | Energy of third ionisation | 2736 kJ.mol ⁻¹ | | Californium |
| Lenntech BV | Standard potential | - 0.3 V (As ³⁺ / As) | | |
| Rotterdamseweg 402 M 2629 HH Delft | Discovered by | The ancients | | Chlorine |
| The Netherlands | | | | Curium |
| tel: +31 15 261 09 00 | | | | Cobalt |
| fax: +31 15 261 62 89 | Arsenic | | | Chromium |
| e-mail: info@lenntech.com | Arsenic appears in three allotropic forms: yellow crystalline solid. It tarnishes rapidly in air, and a | | | Cesium |
| | trioxide. Arsenic is a member of group Va of the The metallic form is brittle, tharnishes and when | | | Copper |
| | odor. The non metallic form is less reactive but | will dissolve when heated with strong or | xidizing acids and alkalis. | Dubnium |
| | Applications | | | Darmstadtium |
| | Arsenic compounds are used in making special t | ypes of glass, as a wood preservative a | and, lately, in the | Dysprosium |
| | semiconductor gallium arsenade, which has the has become an important dopant gas in the mic | Erbium | | |
| | use because it is extremely toxic. During the 18th, 19th, and 20th centuries, a nur | mber of arsenic compounds have been | used as medicines; copper | Einsteinium |
| | acetoarsenite was used as a green pigment kno | wn under many different names. | | Europium |
| | Arsenic in the environment | | | Fluorine |
| | Arsenic can be found naturally on earth in small air, water and land through wind-blown dust and | | | Iron |
| | sources: vulcanoes release about 3000 tonnes per extent of 20.000 tonnes per year, but human ac | er year and microorganisms release vo | platile methylarsines to the | Fermium |
| | year are released by the burning of fossil fuels. | LIVILY IS responsible for much more, ou. | .000 tollies of arsefile per | Francium |
| | Despite its notoriety as a deadly poison, arsenic | | animals, and maybe even | Gallium |
| | for humans, although the necessary intake may | | | Gadolinium |
| | Arsenic is a component that is extremely hard to arsenic is naturally a fairly a mobile component, | , basically means that large concentrati | ions are not likely to | Germanium |
| | appear on one specific site. This is a good thing, wider issue because it easily spreads. Arsenic ca | annot be mobilized easily when it is imm | nobile. Due to human | Hydrogen |
| | activities, mainly through mining and melting, n found on many more places than where they ex | | nobilized and can now be | Helium |
| | A little uncombined arsenic occurs naturally as r | microcrystalline masses, found in Siberi | a, Germany, France, Italy. | Hafnium |
| 931 | Romania and in the USA. Most arsenic is found i realgar, orpiment and enargite. Non is mined as | n conjuction with sulfur in minerals such | h as arsenopyrite (AsFeS), | Mercury |
| 517931 | of other metals, such as copper and lead. World | production of arsenic, in the form of its | s oxide, is around 50.000 | Holmium |
| | tonnes per year, far in excess of that required b and Mexico. World resources of arsenic in coppe | | | Hassium |
| | Health effects of arsenic | | | Iodine |
| | Arsenic is one of the most toxic elements that c | | | Indium |
| | occur on earth naturally in small amounts. Hum Exposure may also occur through skin contact v | with soil or water that contains arsenic. | | Iridium |
| | Levels of arsenic in food are fairly low, as it is n seafood may be high, because fish absorb arser | | | Potassium |

Arsenic exposure may be higher for people that work with arsenic, for people that live in houses that contain conserved wood of any kind and for those who live on farmlands where arsenic-containing pesticides have been applied in the past.

Exposure to inorganic arsenic can cause various health effects, such as irritation of the stomach and intestines, decreased production of red and white blood cells, skin changes and lung irritation. It is suggested that the uptake of significant amounts of inorganic arsenic can intensify the chances of cancer development, especially the chances of development of skin cancer, lung cancer, liver cancer and lymphatic cancer.

A very high exposure to inorganic arsenic can cause infertility and miscarriages with women, and it can cause skin disturbances, declined resistance to infections, heart disruptions and brain damage with both men and women.

Finally, inorganic arsenic can damage DNA.

A lethal dose of arsenic oxide is generally regarded as 100 mg.

Organic arsenic can cause neither cancer, nor DNA damage. But exposure to high doses may cause certain effects to human health, such as nerve injury and stomachaches.

Environmental effects of arsenic

The arsenic cycle has broadened as a consequence of human interference and due to this, large amounts of arsenic end up in the environment and in living organisms. Arsenic is mainly emitted by the copper producing industries, but also during lead and zinc production and in agriculture. It cannot be destroyed once it has entered the environment, so that the amounts that we add can spread and cause health effects to humans and animals on many locations on earth.

Plants absorb arsenic fairly easily, so that high-ranking concentrations may be present in food. The concentrations of the dangerous inorganic arsenics that are currently present in surface waters enhance the chances of alteration of genetic materials of fish. This is mainly caused by accumulation of arsenic in the bodies of plant-eating freshwater organisms. Birds eat the fish that already contain eminent amounts of arsenic and will die as a result of arsenic poisoning as the fish is decomposed in their bodies.

More information on properties of arsenic

Read more on arsenic in water

Information on arsenicosis

Back to the periodic table of elements



Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium

Manganese

Molybdenum Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Neodymiu

Neon

Nickel

Nobelium

Neptunium

Oxygen

Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

ladon

Ruthenium

Sulfur

Antimony

Scandium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium Tellurium

Thorium

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Scientific Facts on Arsenic

Source document: IPCS (2001)

Summary & Details: GreenFacts (2004)

Information on our Three-Level Structure

6. What are the effects of arsenic on the environment?

Water and land-living plants and animals show a wide range of sensitivities to different chemical forms of arsenic. Their sensitivity is modified both by biological factors and by their surrounding physical and chemical environment. In general, inorganic forms of arsenic are more toxic to the environment than organic forms and, among inorganic forms, arsenite is more toxic than arsenate. This is probably because the way in which the various forms are taken up into the body differs and once taken up, they act in different ways in the body. The reason why arsenite is toxic is thought to be because it binds to particular chemical groups - sulfhydryl groups - found on proteins. Arsenate, on the other hand, affects the key energy producing process that take place in all cells.

Arsenic compounds cause short-term and long-term effects in individual plants and animals and in populations and communities of organisms. These effects are evident, for example, in aquatic species at concentrations ranging from a few micrograms to milligrams per litre. The nature of the effects depends on the species and time of exposure. The effects include death, inhibition of growth, photosynthesis and reproduction, and behavioral effects. Environments contaminated with arsenic contain only a few species and fewer numbers within species. If levels of arsenate are high enough, only resistant organisms, such as certain microbes, may be present. <u>More...</u>

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|-------------------------------|--|----------------------------|---|------------------------------------|-----------|--------------|-------|
| About Lenntech | Home > Periodic table > I | Elements > Copper | | | | Elements | To be |
| urnkey plants | Copper - Cu | | | | | Actinium | |
| ilot plants | | | | | | Silver | |
| ontainerized plants | Chemical prop | erties of copper - | Health effects of coppe | r - Environmental effects | of copper | Aluminum | |
| ervices | Atomic number | 29 | i | | | Americium | |
| areer at Lenntech | Atomic mass | 63 | .546 g.mol | | | | |
| nternational Internships | | | ,5 to gillor | | | | |
| eriodic table | Electronegativity according | ording to | 9 | | | | |
| alculators | Density | 8.9 | 9 g.cm ⁻³ at | | | | |
| isitors information | Melting point | 10 | 83 °C | | | | |
| ur partners | 17. 5 | | 95 °C | | | | |
| ontact us | Boiling point | | | OPE | | | |
| | Vanderwaals radius | | 128 nm | 1 | | | |
| Request a | Ionic radius | 0.0 | 096 nm (+1) | | | | |
| Quote ? | Isotopes | 6 | | | | 3. | |
| | Electronic shell | [A | Ar] 3d ¹⁰ 4s ¹ | | | | |
| SHARE ELM. | Energy of first ionisat | ion . 74 | 3.5 kJ.mol ⁻¹ | | | | |
| * | Energy of second ioni | | 46 kJ.mol ⁻¹ | | | | |
| | Energy of Second Ioni | sation 19 | 46 KJ.mol * | | | | |
| Lenntech BV | Standard potential | | 0.522 V (Cu [*] u ²⁺ / Cu) | | | 1 | |
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| 2629 HH Delft The Netherlands | Discovered by | *** | e ancients | | | Curium | |
| tel: +31 15 261 09 00 | Copper | | | | | Cobalt | |
| fax: +31 15 261 62 89 | | | d cubic crystalline structure. It r | | | Chromium | |
| e-mail: info@lenntech.com | malleable, ductile, and a | n extremely good con | um, due to its band structure, so iductor of both heat and electric | ity. It is softer than zinc and ca | n be | Cesium | |
| | low chemical reactivity. | In moist air it slowly for | Ib of the <u>periodic table</u> , togethe orms a greenish surface film cal | | | Copper | |
| | metal from further attac | k. | | | | Dubnium | |
| * | Applications | | | | | Darmstadtium | |
| 200 | | | 50%); construction, such as roof d alloys (5%). The main long es | | | Dysprosium | |
| | | | nich was strong enough to make pronickel, which was the preferr | | | Erbium | |
| | coins. Copper is ideal for electr | ical wiring because it | is easily worked, can be drawn | into fine wire and has a high | | Einsteinium | |
| | electrical conductivity. | * * * | | | | Europium | |
| | Copper in the environme | ent | | | | Fluorine | |
| | | | rs naturally in the environment mans widely use copper. For inst | | es | Iron | |
| | | roduction of copper ha | as lifted over the last decades. | | | Fermium | |
| | | | is basically means that more ar | nd more copper ends up in the | | Francium | |
| | environment. Rivers are | depositing sludge on | their banks that is contaminated the air, mainly through release | d with copper, due to the dispos | al of | Gallium | |
| | fuels. Copper in air will r | emain there for an en | ninent period of time, before it a ay also contain large quantities | settles when it starts to rain. It | | Gadolinium | |
| | has settled. | | -,se contain large quantities | | | Germanium | |
| pr # . | | | by both natural sources and hur tion, forest fires and sea spray. | | | Hydrogen | |
| | that contribute to copper production and phosphat | release have already | been named. Other examples | are mining, metal production, v | vood | Helium | |
| | Because copper is releas | ed both naturally and | through human activity it is ver | | nt. | Hafnium | |
| | The second secon | | nd to either water sediment or | | | Mercury | |
| | | gest threat to human I | health. Usually water-soluble co | | | Holmium | |
| | | - op | | | | Hassium | |
| | tons, which are expected | to last for only anoth | lion tons a year and exploitable per 25 years. About 2 million ton | ns a year are reclaimed by | ū | Iodine | |
| | together account for aro | | osits in Chile, Indonesia, USA, as copper. The main ore is a yell | | | Indium | |
| | chalcopyrite (CuFeS2). | | | | | Iridium | |
| | Health effects of co | | | | | | |

Copper can be found in many kinds of food, in drinking water and in air. Because of that we absorb eminent quantities of copper each day by eating, drinking and breathing. The absorption of copper is necessary, because copper is a trace element that is essential for human health. Although humans can handle proportionally large concentrations of copper, too much copper can still cause eminent health problems.

Copper concentrations in air are usually quite low, so that exposure to copper through breathing is negligible. But people that live near smelters that process copper ore into metal, do experience this kind of exposure.

People that live in houses that still have copper plumbing are exposed to higher levels of copper than most people, because copper is released into their drinking water through corrosion of pipes.

Occupational exposure to copper often occurs. In the working environment, copper contagion can lead to a flu-like condition known as metal fever. This condition will pass after two days and is caused by over sensitivity.

Effects

Long-term exposure to copper can cause irritation of the nose, mouth and eyes and it causes headaches, stomachaches, dizziness, vomiting and diarrhea. Intentionally high uptakes of copper may cause liver and kidney damage and even death. Whether copper is carcinogenic has not been determined yet.

There are scientific articles that indicate a link between long-term exposure to high concentrations of copper and a decline in intelligence with young adolescents. Whether this should be of concern is a topic for further investigation.

Industrial exposure to copper fumes, dusts, or mists may result in metal fume fever with atrophic changes in nasal mucous membranes. Chronic copper poisoning results in Wilson's Disease, characterized by a hepatic cirrhosis, brain damage, demyelization, renal disease, and copper deposition in the cornea.

Environmental effects of copper

When copper ends up in soil it strongly attaches to organic matter and minerals. As a result it does not travel very far after release and it hardly ever enters groundwater. In surface water copper can travel great distances, either suspended on sludge particles or as free ions.

Copper does not break down in the environment and because of that it can accumulate in plants and animals when it is found in soils. On copper-rich soils only a limited number of plants has a chance of survival. That is why there is not much plant diversity near copper-disposing factories. Due to the effects upon plants copper is a serious threat to the productions of farmlands. Copper can seriously influence the proceedings of certain farmlands, depending upon the acidity of the soil and the presence of organic matter. Despite of this, copper-containing manures are still applied.

Copper can interrupt the activity in soils, as it negatively influences the activity of microorganisms and earthworms. The decomposition of organic matter may seriously slow down because of this.

When the soils of farmland are polluted with copper, animals will absorb concentrations that are damaging to their health. Mainly sheep suffer a great deal from copper poisoning, because the effects of copper are manifesting at fairly low concentrations.

Back to periodic chart

Recommended daily intake of copper



Krypto

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium

Manganese

Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium

Oxygen

Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Praseodymium

Platinum

Plutonium

Radium

Rubidium

Rhenium

Rutherfordium Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Scandium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium Thorium

Birds

No data are available on the toxicity of copper to avian wildlife. All studies with birds and copper use domestic chickens, ducks, or turkeys (Table 6). Copper, however, may indirectly affect avian wildlife by curtailing certain prey species. Winger et al. (1984), for example, show that apple snails (*Pomacea paludosa*) are not only extremely susceptible to copper (LC50 of 24 to 57 µg/L in 96 h; immatures most sensitive), but are the primary food of the snail kite (*Rostrhamus sociabilis*), an endangered species. The decline of the apple snail in southern Florida coincided with the use of copper-diquat to control hydrilla aquatic weeds (*Hydrilla verticillata*), with serious implications for the snail kite (Winger et al. 1984).

In the domestic chicken, adverse effects of copper occur in chicks fed diets containing 350 mg Cu/kg ration for 25 days (reduced weight gain) and in adults given a dietary equivalent of more than 28 mg Cu/kg BW (Table 6). Chicks fed diets of 500 mg Cu/kg ration show damage to the gizzard lining; damage effects are attributed to the shedding of gizzard glandular cells into the keratin-like koilin layer, disrupting koilin production (Bremner 1979). Copper-induced gizzard histopathology in growing chicks is not reversed by zinc or vitamins B₁₂ or E (Poupoulis and Jensen 1976). Supplementing chick diets with copper did not prove markedly advantageous (Poupoulis and Jensen 1976), provided that normal rations had about 4 mg Cu/kg and adequate iron (Carlton and Henderson 1964b). Unlike mammals, chicks fed copper-supplemented diets do not have elevated copper concentrations in liver or signs of liver damage (Bremner 1979). Broiler hens housed on slats made of lumber pressure-treated with chromated copper arsenate showed severe foot-pad dermatitis and excessive mortality after 17 weeks; however, arsenic and cresylic acid—not copper—may be the responsible agents (Sander et al. 1994).

Ducklings (*Anas* spp.), unlike chicks, accumulate copper in livers when fed diets supplemented with high loadings of copper (Wood and Worden 1973). Domesticated mallards show a dose-time dependent increase in copper liver concentrations, with a maximum concentration of 254 mg Cu/kg DW liver (Table 6). Mallards seem to prefer drinking water containing 100 mg Cu/L over distilled water (Table 6); however, these birds were molting and this may have influenced their response because trace mineral requirements rise during molting (Rowe and Prince 1983).

In turkeys, natural diets with as much as 800 mg Cu/kg ration have no adverse effects on growth or survival. But purified diets are toxic to turkeys in three weeks, and purified diets that contain as little as 50 mg Cu/kg ration produce adverse effects (Waibel et al. 1964). Turkeys fed purified diets with supplemented copper show a dose-dependent increase in mortality and decrease in growth; these effects are attributed to a copper-accelerated dietary deterioration (Supplee 1964). Turkey growth and survival are acceptable when fed purified diets supplemented with as much as 800 mg Cu/kg ration provided that effective levels of added antioxidant (0.02% ethoxyquin) and stabilized sources of vitamins A and D are present (Supplee 1964).

Mammals

Wilson's disease is the only naturally occurring neuropathological condition in humans and other mammals in which copper poisoning is implicated. People with Wilson's disease have severe pathological changes in the brain, especially in the basal ganglia, and in the liver; pathology is associated with excess copper in tissues (Doherty et al. 1969). Copper concentrations in tissues from children that die from Wilson's disease are as much as 2,217 mg/kg DW in liver and 1,245 mg/kg DW in kidney (Table 7). Long-term exposure of humans to copper dust irritates the nose, eyes, and mouth and causes headaches, dizziness, nausea, and diarrhea (USEPA 1980; ATSDR 1990). Drinking water that contains higher than normal concentrations of copper may cause vomiting, diarrhea, stomach cramps, nausea, and greenish or bluish stools and saliva. Intentionally high intakes of copper may result in liver and kidney damage, and sometimes death, especially in children. The seriousness of the effects of copper is expected to increase with increasing dose and duration of exposure (USEPA 1980; ATSDR 1990). Human tissues exposed directly to copper or copper salts will suffer adverse effects because of copper absorption. This is the case for copper bracelets on sweaty skin, for certain intrauterine devices, and for copper dental fillings (USEPA 1980). In monkeys, copper used as dental fillings in deciduous teeth causes more severe pulp damage than did other materials studied (USEPA 1980).

Mammals and birds are 100-1,000 times more resistant to copper than other animals (Schroeder et al. 1966). But excessive dietary intakes of copper by 20- to 50-fold over normal levels may have serious effects in mammals. Depending on the species, growth and food intake may be reduced, anemia may develop, and liver,

kidney, brain, and muscle may degenerate, often resulting in death (Bremner 1979; ATSDR 1990). Copper poisoning in mammals may result from consumption of plants treated with copper-containing pesticides, from the veterinary use of copper sulfate to control helminthiasis and infectious pododermatitis in cattle and sheep, and from the ingestion of contaminated soils and vegetation near copper mining and refining operations (NAS 1977). Emissions from copper mines and smelters are often associated with deaths of horses, cows, and sheep; pasture lands, in some cases, are fit for grazing only after heavy rains (Hutchinson 1979).

Ruminant mammals are significantly more sensitive to copper than nonruminant mammals and poultry (Schroeder et al. 1966; NAS 1977). Signs of copper poisoning in ruminants include vomiting, excessive salivation, abdominal pain, diarrhea with greenish-tinted feces, pathology of internal organs, elevated copper concentrations in liver, altered enzyme activities in liver and serum, and collapse and death within 24 to 48 h (NAS 1977). Young calves may develop copper toxicosis at relatively low copper intakes, especially when receiving milk-based diets; goats, however, seem resistant to copper toxicosis (Bremner 1979). Among ruminants, domestic sheep are particularly susceptible to copper insult from grazing on pastures treated with copper-containing fungicides and molluskicides or from inadvertently consuming diets specially formulated for pigs and which contain large amounts of copper as a swine growth stimulant (Todd 1969; Bremner 1979).

Chronic copper poisoning in domestic sheep is first characterized by a period of passive accumulation of copper in the tissues. This period varies from a few weeks to more than a year. During this time the animal appears outwardly normal although the liver may contain more than 1,000 mg Cu/kg DW and plasma activities of aspartate transaminase, sorbitol dehydrogenase, lactic dehydrogenase, and arginase increase, indicating that liver damage has occurred. During the last few weeks of the passive phase, and prior to the so-called toxic phase, liver histopathology of parenchymal cells and copper-containing Kupffer cells occurs. The toxic phase, which is an acute illness and referred to as the hemolytic crisis, usually results in death 2-4 days later. During this phase sheep refuse to eat but have an excessive thirst. The eyes are usually sunken. The venous blood is chocolate colored. The liver is jaundiced. The kidneys are completely gorged with hemoglobin breakdown products and the medulla and cortex are black. The spleen is enlarged, with the parenchyma a deep brown to black color. The onset of these signs in sheep is associated with liberation of copper from the liver and a massive increase in blood copper concentrations. The increased blood copper concentrations lead to an increase in blood methemoglobin and a sudden fall in the erythrocyte glutathione level immediately followed by massive hemolysis and kidney damage, leading to uremia and death. At the time of crisis, elevated serum creatine phosphokinase activity suggests that muscle cell membranes are affected, and elevated serum glutamic oxaloacetate transaminase (SGOT) and lactic dehydrogenase activities indicate progressive liver necrosis (Doherty et al. 1969; Todd 1969; Thompson and Todd 1974; Bremner 1979). It is emphasized that (1) blood copper status and liver function in sheep experimentally poisoned with copper sulfate are linked to elevated SGOT activities 1 to 6 weeks in advance of obvious external signs (MacPherson and Hemingway 1969); (2) copper chloride is 2 to 4 times more toxic to sheep than copper sulfate is (NAS 1977); and (3) the use of copper-enriched feeding stuffs increases the risk of chronic copper poisoning in sheep fed purified rations (Froslie et al. 1983). Also, sheep that accumulate higher than normal amounts of copper in the liver (i.e., 1,900 mg Cu/kg DW) are more severely affected by lupinosis (acute liver atrophy due to poisoning by ingestion of plants of Lupinus spp.) than sheep with normal (40 mg/kg DW) concentrations of copper in the liver (Gardiner 1967).

Copper toxicosis in lambs of domestic sheep occurs at dietary concentrations between 8 and 60 mg Cu/kg ration. The wide range of dietary concentrations is a function of copper availability. Availability, in turn, is influenced by dietary composition, genetic influence, age, breed, sex, physiological state, and interactions with other dietary constituents including iron, zinc, and molybdenum (Bremner 1979). Chronic copper poisoning in lambs occurs at dietary levels as low as 27 mg Cu/kg DW ration (Buckley and Tait 1981). During the passive phase, lambs—like adults—have normal plasma copper concentrations and seem outwardly unaffected. Unlike adults, copper accumulates in livers of lambs during a shorter period (several weeks to months vs. months to years). Signs of hemolytic crisis and death within a few days are similar for both adults and lambs. Elevated plasma aspartate aminotransferase (AAT) activity in lambs—up to 10 times higher than controls—occurs 4 to 8 weeks before the hemolytic crisis (Buckley and Tait 1981) and strongly indicates a need for more research on the usefulness of AAT and other enzymes as early indicators of copper stress. A recommended treatment for lambs diagnosed with chronic copper poisoning is 20 mL of a mixture containing 100 mg of ammonium molybdate and 1 g of sodium sulfate administered orally 5 days weekly (Doherty et al. 1969).

| Home | Applications | Processes | Systems | Products | Library | Languages | |
|---------------------------------------|--|--|--|--|-------------------|--------------|--|
| bout Lenntech | Home > Periodic table > | Elements > Nickel | | | | Elements | |
| urnkey plants | Nickel - Ni | | | | | Actinium | |
| lot plants | ř. | | | | | Silver | |
| ntainerized plants | Chemical pro | operties of nickel | - Health effects of nickel | - Environmental effect | s of nickel | Aluminum | |
| ervices | Atomic number | | 28 | | | Americium | |
| reer at Lenntech | Atomic mass | | 58.71 g.mol ⁻¹ | | | Argon | |
| ternational Internships | Electronegativity acc | ording to Pauling | 1.8 | | | Arsenic | |
| riodic table | | ording to rading | | | | Astatine | |
| Iculators | Density | | 8.9 g.cm ⁻³ at 20°C | | | Gold | |
| sitors information | Melting point | | 1453 °C | | | Boron | |
| ır partners | Boiling point | | 2913 °C | | | Barium | |
| entact us | Vanderwaals radius | | 0.124 nm | | | Beryllium | |
| | Ionic radius | | 0.069 nm (+2); 0.06 nm (- | +3) | | Bohrium | |
| Request a | Isotopes | | 10 | | 100 x 172 ft 52 | Bismuth | |
| Quote ? | 20 (SE) 150 (No. | | | | Tar Co | Berkelium | |
| | Electronic shell | | [Ar] 3d ⁸ 4s ² | | | Bromine | |
| SHARE BEM | Energy of first ionisa | ition | 735 kJ.mol ⁻¹ | | | Carbon | |
| | Energy of second ion | isation | 1753 kJ.mol ⁻¹ | | | Calcium | |
| | Energy of third ionis | ation | 3387 kJ.mol ⁻¹ | | | Cadmium | |
| | , | | | | | Cerium | |
| Lenntech BV | Standard potential | | - 0.25 V | | | Californium | |
| Rotterdamseweg 402 M 2629 HH Delft | Discovered by | | Alex Constedt 1751 | | | Chlorine | |
| The Netherlands | | | | | Y | Curium | |
| tel: +31 15 261 09 00 | Nickel | | | | | Cobalt | |
| fax: +31 15 261 62 89 | | nard, malleable, and du | actile metal. It is of the iron gro | up and it takes on a high polis | n. It is a | Cesium | |
| e-mail: info@lenntech.com | fairly good conductor of valences. It also forms | heat and electricity. In a number of complex of | its familiar compounds nickel is empounds. Most nickel compour | s bivalent, although it assumes nds are blue or green. Nickel o | other issolves | Copper | |
| | hydrogen. | t, like fron, becomes pa | ssive when treated with nitric a | icia. Finely divided nicker adso | rbs | Dubnium | |
| | Applications | | | | | Darmstadtium | |
| | | | alloys. Nickel alloys are charac | | | Dysprosium | |
| | steel, whose composition | on can vary but is typica | f the nickel consumed in the We illy <u>iron</u> with around 18% <u>chrom</u> | nium and 8% nickel. 12 % of a | | Erbium | |
| | rechargeable batteries, | catalysts and other che | maining 23% of consumption is micals, coinage, foundry produ | cts, and plating. | | Einsteinium | |
| | is used in gas turbines a | and rocket engines. Mor | ire. It resist corrosion even at hele is an alloy of nickel and copp | per (e.g. 70% nickel, 30% cop | per with | Europium | |
| | traces of iron, mangane for propeller shaft in bo | | s not only hard but can resist co nts. | rrosion by sea water, so that i | t is ideal | Fluorine | |
| | Nickel in the environme | nt | | | | Iron | |
| x . | Most nickel on Earth is i | naccessible because it i | s locked away in the planet's iro | on-nickel molten core, which is | 10 % | Fermium | |
| | nickel. The total amoun | t of nickel dissolved in t | he sea has been calculated to be swhy coal and oil contain consi | e around 8 billion tons. Organ | ic matter | Francium | |
| | soil can be as low as 0.2 | 2 ppm or as high as 450 | ppm in some clay and loamy sential component of some enzyr | oils. The average is around 20 | ppm. | Gallium | |
| | nickel is tea which has 7 Nickel occurs combined | | es. e, with <u>arsenic</u> in the mineral ni | ccolite, and with arsenic and s | ulphur in | Gadolinium | |
| | | | tracted are iron-nickel sulphide ba, Canada and South Africa. Ar | | | Germanium | |
| | and easily workable res | erves will last at least : | 150 years. | | | Hydrogen | |
| | Health effects of n | ickel | | | | Helium | |
| | | | ment only at very low levels. Honckel is the use as an ingredien | | | Hafnium | |
| | can be found in commo | | | | | Mercury | |
| | | | | | | Holmium | |
| | | | ickel. Chocolate and fats are kn | | | Hassium | |
| | accumulate nickel and a | is a result the nickel up | quantities of vegetables from p take from vegetables will be en | | | Iodine | |
| | uptake through their lur | 70 20 1 | - | d or smoking digarettes. Clii- | contact | Indium | |
| | with nickel-contaminate | d soil or water may als | g air, <u>drinking water</u> , eating foo o result in nickel exposure. In s | | | Iridium | |
| | when the uptake is too | gir ic can be a danger | to numan neath. | | | Potassium | |

- Sickness and dizziness after exposure to nickel gas
- Lung embolism
- Respiratory failure
- Birth defects
 Asthma and chronic bronchitis
- Allergic reactions such as skin rashes, mainly from jewelry
- Heart disorders

Nickel fumes are respiratory irritants and may cause pneumonitis. Exposure to nickel and its compounds may result in the development of a dermatitis known as "nickel itch" in sensitized individuals. The first symptom is usually itching, which occurs up to 7 days before skin eruption occurs. The primary skin eruption is erythematous, or follicular, which may be followed by skin ulceration. Nickel sensitivity, once acquired, appears to persist indefinitely.

Carcinogenicity- Nickel and certain nickel compounds have been listed by the National Toxicology Program (NTP) as being reasonably anticipated to be carcinogens. The International Agency for Research on Cancer (IARC) has listed nickel compounds within group 1 (there is sufficient evidence for carcinogenicity in humans) and nickel within group 28 (agents which are possibly carcinogenic to humans). OSHA does not regulate nickel as a carcinogen. Nickel is on the ACGIH Notice of Intended Changes as a Category A1, confirmed human carcinogen.

Effects of nickel on the environment

Nickel is released into the air by power plants and trash incinerators. It will than settle to the ground or fall down after reactions with raindrops. It usually takes a long time for nickel to be removed from air. Nickel can also end up in surface water when it is a part of <u>wastewater</u> streams.

The larger part of all nickel compounds that are released to the environment will adsorb to sediment or soil particles immobile as a result. In acidic ground however, nickel is bound to become more mobile and it will often rinse out to the groundwater

There is not much information available on the effects of nickel upon organisms other than humans. We do know that high nickel concentrations on sandy soils can clearly damage plants and high nickel concentrations in surface waters can diminish the growth rates of algae. Micro organisms can also suffer from growth decline due to the presence of nickel, but they usually develop resistance to nickel after a while.

For animals nickel is an essential foodstuff in small amounts. But nickel is not only favorable as an essential element: it can also be dangerous when the maximum tolerable amounts are exceeded. This can cause various kinds of cancer on different sites within the bodies of animals, mainly of those that live near refineries.

Nickel is not known to accumulate in plants or animals. As a result nickel will not bio magnify up the food chain.

Now check out our page on nickel and water

Back to **periodic chart**

Recommended daily intake of nickel

Refractory Metals Tantalum, Tungsten, Molybdenum, Niobium. Extensive Inventory. www.eagkellovs.com

BryCoat Metallurgical Coa Coating for Improved Performance of Metal Components. www.brycoat.com

Aluminum & Zinc Castings Good looking, strong parts Low cost graphite mould technology www.grinexas AdChoices ▶

Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium Manganese

Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Neon

Nobelium

Neptunium

Oxygen

Osmium

Protactinium

Lead

Palladium

Promethium

Praseodymium

Platinum

Plutonium

Radium

Rubidium Rhenium

Rutherfordium

Roentgenium

Rhodium

Ruthenium

Sulfur

Antimony

Scandium

Seaborgium

Silicon

Strontium

Tantalum

Terbium

Technetium Tellurium

Thorium

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| ırnkey plants | Chromium - Cr | | | | | Actinium |
| ot plants | | | | | | Silver |
| ntainerized plants | Chemical properties of ch | romium - Hea | Ith effects of chromiu | m - Environmental effe | ects of chromium | Aluminum |
| ervices | Atomic number | 24 | | | | Americium |
| reer at Lenntech | Atomic mass | 51.996 g.m | nol ⁻¹ | | 2 | Argon |
| ternational Internships | Electronegativity | 1.6 | | | | Arsenic |
| riodic table | 50000000 0000000 - 0000000 - | | 3 | | | Astatine |
| Iculators | Density | 7.19 g.cm ⁻ | at 20°C | | | Gold |
| itors information | Melting point | 1907 °C | * | | | Boron |
| r partners | Boiling point | 2672 °C | | 6.50 | | Barium |
| ntact us | Vanderwaals radius | 0.127 nm | | | * | Beryllium |
| - | Ionic radius | 0.061 nm (| +3); 0.044 nm (+6) | | | Bohrium |
| Request a | Isotopes | 6 | | and & | | Bismuth |
| Quote ? | | | .J | Sales . | | Berkelium |
| | Electronic shell | [Ar] 3d ⁵ 4 | 1 5 - | | | Bromine |
| SHARE ILE | Energy of first ionisation | 651.1 kJ.m | ol ⁻¹ | | | Carbon |
| | Energy of second ionisation | 1590.1 kJ.r | mol ⁻¹ | X. | | Calcium |
| | Energy of first ionisation | 2987 kJ.mo | ol ⁻¹ | | | Cadmium |
| Lenntech BV | Standard potential | - 0.71 V (C | 3+ (0 -) | | | Californium |
| | (c) 5000000000000 ▼ No.000000000000 | 2200 | | | | Chlorine |
| Rotterdamseweg 402 M 2629 HH Delft The Netherlands | Discovered by | Vaughlin in | 1797 | | | Curium |
| tel: +31 15 261 09 00 | Chromium | | | | | Cobalt |
| fax: +31 15 261 62 89 | Chromium is a lustrous, brittle, l | ard metal. Its co | lour is silver-gray and it can | he highly polished. It does no | nt. | Chromium |
| -mail: info@lenntech.com | tarnish in air, when heated it bor immediately produces a thin oxid | ns and forms the | green chromic oxide. Chrom | nium is unstable in oxygen, it | λ. | Cesium |
| | Applications | de layer that is in | ipermeable to oxygen and pr | rotects the metal below. | | Copper |
| | Chromium main uses are in alloy | | | | | Dubnium |
| đ | practing was once widely used to give steel a poinsned silvery mirror coating. Enromium is used in mectaning to impart corrosion resistance and a shiny finish; as dyes and paints, its salts colour glass an emerald green and it is used to produce synthetic rubies; as a catalyst in dyeing and in the tanning of leather; to make molds for the firing of bricks. Chromium (IV) oxide (CrO-) is used to manufacture mannet; take | | | | | Darmstadtium |
| | | | | | | Dysprosium |
| | Chromium in the environment | | | | | Erbium |
| | Chromium is mined as chromite | | | | a, | Einsteinium |
| | | | | | | Europium |
| | USA. | | | | | Fluorine |
| | Health effects of chromiu | m | | | | Iron |
| | People can be exposed to chrom chromium or chromium compou | nds. The level of | chromium in air and water is | generally low. In drinking w | ater | Fermium |
| | the level of chromium is usually chromium(IV); hexavalent chro | mium. For most p | eople eating food that contain | ins chromium(III) is the mair | | Francium |
| | route of chromium uptake, as cl grains. Various ways of food pre | paration and stor | age may alter the chromium | | | Gallium |
| | stores in steel tanks or cans chr | | | | \$ 8 | Gadolinium |
| | Chromium(III) is an essential netabolisms and diabetes. But | | | | of | Germanium |
| | instance skin rashes. | No. of the Control of | | | | Hydrogen |
| | Chromium(VI) is a danger to hu People who smoke tobacco also | | | | | Helium |
| | Chromium(VI) is known to caus | | | | ause | Hafnium |
| | allergic reactions, such as skin r nosebleeds. | | 3 | iuse nose irritations and | | Mercury |
| | Other health problems that are | caused by chromi | um(VI) are: | | | Holmium |
| | - Skin rashes - Upset stomachs and ulcers | | | | | Hassium |
| | Respiratory problems Weakened immune systems | | | | | Iodine |
| | Kidney and liver damage Alteration of genetic material | x | | | | Indium |
| | - Lung cancer - Death | | | | | Iridium |
| | | | | | | Potassium |

Krypton

Lanthanum Lithium

Lutetium Mendelevium Magnesium Manganese Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium Neodymium

Neon

Nickel

Nobelium Neptunium

Oxygen

Osmium

the hexavalent form on the skin may include ulcerations, dermatitis, and allergic skin reactions. Inhalation of hexavalent chromium compounds can result in ulceration and perforation of the mucous membranes of the nasal septum, irritation of the pharynx and larynx, asthmatic bronchitis, bronchospasms and edema. Respiratory symptoms may include coughing and wheezing, shortness of breath, and nasal itch.

Carcinogenicity- Chromium and most trivalent chromium compounds have been listed by the National Toxicology Program (NTP) as having inadequate evidence for carcinogenicity in experimental animals. According to NTP, there is sufficient evidence for carcinogenicity in experimental animals for the following hexavalent chromium compounds; calclum chromate, and calclum chromium metal and its trivalent compounds within Group 3 (The agent is not classifiable as to its carcinogenicity to humans.) Chromium is not regulated as a carcinogen by OSHA (29 CFR 1910 Subpart Z). ACGIH has classified chromium metal and trivalent chromium compounds as A4,not classifiable as a human carcinogen.

Environmental effects of chromium

There are several different kinds of chromium that differ in their effects upon organisms. Chromium enters the air, water and soil in the chromium (III) and chromium(V1) form through natural processes and human activities.

The main human activities that increase the concentrations of chromium (III) are steal, leather and textile manufacturing. The main human activities that increase chromium(VI) concentrations are chemical, leather and textile manufacturing, electro painting and other chromium(VI) applications in the industry. These applications will mainly increase concentrations of chromium in water. Through coal combustion chromium will also end up in air and through waste disposal chromium will end up in soils.

Most of the chromium in air will eventually settle and end up in waters or soils. Chromium in soils strongly attaches to soil particles and as a result it will not move towards groundwater. In water chromium will absorb on sediment and become immobile. Only a small part of the chromium that ends up in water will eventually discolve.

Chromium(III) is an essential element for organisms that can disrupt the sugar metabolism and cause heart conditions, when the daily dose is too low. Chromium(VI) is mainly toxic to organisms. It can alter genetic materials and cause cancer.

Crops contain systems that arrange the chromium-uptake to be low enough not to cause any harm. But when the amount of chromium in the soil rises, this can still lead to higher concentrations in crops. Acidification of soil can also influence chromium uptake by crops. Plants usually absorb only chromium(III). This may be the essential kind of chromium, but when concentrations exceed a certain value, negative effects can still occur.

Chromium is not known to accumulate in the bodies of fish, but high concentrations of chromium, due to the disposal of metal products in surface waters, can damage the gills of fish that swim near the point of disposal. In animals chromium can cause respiratory problems, a lower ability to fight disease, birth defects, infertility and tumor formation.

Read more on chromium in water

Back to the periodic table of elements.

Recommended daily intake of chromium

<u>Download Google Chrome</u> Searching is fast and easy with Google's web browser. <u>www.google.com/chrome</u>

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Phosphorus

Protactinium Lead

Promethium

Polonium

Praseodymium

Platinum

Radium

Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon Ruthenium

Sulfur

ntimony

Scandium

Selenium

Seaborgium

....

Samarium

Tin

Strontium

antalum

Terbium

Technetium Tellurium

Thorium



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Review article

Chromium toxicity in plants

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Abstract

Due to its wide industrial use, chromium is considered a serious environmental pollutant. Contamination of soil and water by chromium (Cr) is of recent concern. Toxicity of Cr to plants depends on its valence state: Cr(VI) is highly toxic and mobile whereas Cr(III) is less toxic. Since plants lack a specific transport system for Cr, it is taken up by carriers of essential ions such as sulfate or iron. Toxic effects of Cr on plant growth and development include alterations in the germination process as well as in the growth of roots, stems and leaves, which may affect total dry matter production and yield. Cr also causes deleterious effects on plant physiological processes such as photosynthesis, water relations and mineral nutrition. Metabolic alterations by Cr exposure have also been described in plants either by a direct effect on enzymes or other metabolites or by its ability to generate reactive oxygen species which may cause oxidative stress. The potential of plants with the capacity to accumulate or to stabilize Cr compounds for bioremediation of Cr contamination has gained interest in recent years.

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Keywords: Chromium; Toxicity; Plants; Crops; Cr(III); Cr(VI); Photosynthesis; Phytoremediation; Bioremediation; Uptake; Translocation; Reactive Oxygen Species; Oxidative stress; Heavy metals

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URL: http://www.geocities.com/arunshank (A.K. Shanker).

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ATSDR Agency for Toxic Substances & Disease Registry ToxFAQsTM for Barium

(Bario (/es/toxfaqs/es_tfacts24.html))

August 2007

CAS#: 7440-39-3

[] (/tfacts24.pdf) PDF Version, 44 KB (/tfacts24.pdf)

This fact sheet answers the most frequently asked health questions (FAQs) about barium and barium compounds. For more information, call the ATSDR Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because these substances may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

Highlights

Exposure to barium occurs mostly in the workplace or from drinking contaminated water. Ingesting drinking water containing levels of barium above the EPA drinking water guidelines for relatively short periods of time can cause gastrointestinal disturbances and muscle weakness. Ingesting high levels for a long time can damage the kidneys. Barium and barium compounds have been found in at least 798 of the 1,684 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is barium?

Barium is a silvery-white metal which exists in nature only in ores containing mixtures of elements. It combines with other chemicals such as sulfur or carbon and oxygen to form barium compounds.

Barium compounds are used by the oil and gas industries to make drilling muds. Drilling muds make it easier to drill through rock by keeping the drill bit lubricated. They are also used to make paint, bricks, ceramics, glass, and rubber.

Barium sulfate is sometimes used by doctors to perform medical tests and to take x-rays of the gastrointestinal tract.

What happens to barium when it enters the environment?

- Barium gets into the air during the mining, refining, and production of barium compounds, and from the burning of coal and oil.
- The length of time that barium will last in air, land, water, or sediments depends on the form of barium released.
- Barium compounds, such as barium sulfate and barium carbonate, which do not dissolve well in water, can last a long time in the environment.

- Barium compounds, such as barium chloride, barium nitrate, or barium hydroxide, that dissolve easily in water usually do not last in these forms for a long time in the environment. The barium in these compounds that is dissolved in water quickly combines with sulfate or carbonate that are naturally found in water and become the longer lasting forms (barium sulfate and barium carbonate).
- · Fish and aquatic organisms can accumulate barium.

How might I be exposed to barium?

- Ingesting small amounts present in your food and water or breathing air containing very low levels of barium.
- Living in areas with unusually high natural levels of barium in the drinking water.
- Working in a job that involves barium production or use.
- Living or working near waste sites where barium has been disposed of.

How can barium affect my health?

The health effects of the different barium compounds depend on how well the compound dissolves in water or in the stomach contents. Barium compounds that do not dissolve well, such as barium sulfate, are not generally harmful.

Barium has been found to potentially cause gastrointestinal disturbances and muscular weakness when people are exposed to it at levels above the EPA drinking water standards for relatively short periods of time. Some people who eat or drink amounts of barium above background levels found in food and water for a short period may experience vomiting, abdominal cramps, diarrhea, difficulties in breathing, increased or decreased blood pressure, numbness around the face, and muscle weakness. Eating or drinking very large amounts of barium compounds that easily dissolve can cause changes in heart rhythm or paralysis and possibly death. Animals that drank barium over long periods had damage to the kidneys, decreases in body weight, and some died.

How likely is barium to cause cancer?

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified barium as to its carcinogenicity. The EPA has determined that barium is not likely to be carcinogenic to humans following ingestion and that there is insufficient information to determine whether it will be carcinogenic to humans following inhalation exposure.

How can barium affect children?

We do not know whether children will be more or less sensitive than adults to barium toxicity. A study in rats that swallowed barium found a decrease in newborn body weight; we do not know if a similar effect would be seen in humans.

How can families reduce the risks of exposure to barium?

• The greatest potential source of barium exposure is through food and drinking water. However, the amount of barium in foods and drinking water are typically too low to be of concern.

Is there a medical test to determine whether I've been exposed to barium?

There is no routine medical test to determine whether you have been exposed to barium. Doctors can measure barium in body tissues and fluids, such as bones, blood, urine, and feces, using very complex instruments. These tests cannot be used to predict the extent of the exposure or potential health effects.

The geometric mean barium level measured in the U.S. general population aged 6 and older is reported by the Centers for Disease Control and Prevention (CDC) as 1.44 ig/g creatinine (measured in urine).

Has the federal government made recommendations to protect human health?

The EPA has set a limit of 2.0 milligrams of barium per liter of drinking water (2.0 mg/L), which is the same as 2 ppm.

The Occupational Safety and Health Administration (OSHA) has set Permissible Exposure Limits (PELs) of 0.5 milligrams of soluble barium compounds per cubic meter of workplace air (0.5 mg/m 3) for 8 hour shifts and 40 hour work weeks. The OSHA limits for barium sulfate dust are 15 mg/m 3 of total dust and 5 mg/m 3 for respirable fraction.

The National Institute for Occupational Safety and Health (NIOSH) has set Recommended Exposure Limits (RELs) of 0.5 mg/m³ for soluble barium compounds. The NIOSH has set RELs of 10 mg/m³ (total dust) for barium sulfate and 5 mg/m³ (respirable fraction).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2007. <u>Toxicological Profile for Barium and Compounds (/ToxProfiles/TP.asp?id=327&tid=57)</u> (*Update*). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

If you have questions or concerns, please contact your community or state health or environmental quality department or:

For more information, contact:

Agency for Toxic Substances and Disease Registry Division of Toxicology and Environmental Medicine 1600 Clifton Road NE, Mailstop F-62

Atlanta, GA 30333

Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)

Fax: 1-770-488-4178

Email: cdcinfo@cdc.gov (mailto:cdcinfo@cdc.gov)

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Information line and technical assistance:

Phone: 888-422-8737 FAX: (770)-488-4178

To order toxicological profiles, contact:

National Technical Information Service 5285 Port Royal Road Springfield, VA 22161

Phone: 800-553-6847 or 703-605-6000

Disclaimer

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- Page last reviewed: March 3, 2011
- Page last updated: March 3, 2011
- Content source: Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/)

Agency for Toxic Substances and Disease Registry, 4770 Buford Hwy NE, Atlanta, GA 30341

Contact CDC: 800-232-4636 / TTY: 888-232-6348



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|--|--|---|--|--|--------------|--------------|-----|
| About Lenntech | <u>Home</u> > <u>Periodic table</u> > <u>Ele</u> | ments > Beryllium | | | | Elements | |
| Turnkey plants | Beryllium - Be | 5 | 3 | | | Actinium | |
| Pilot plants | | | H m | * | | Silver | |
| Containerized plants | Chemical properties | of beryllium - I | Health effects of beryllium | n - Environmental effects | of beryllium | Aluminum | |
| Services | | | | | | Americium | |
| Career at Lenntech | Atomic number | | 4 | | | Argon | |
| International Internships | Atomic mass | | 9.01218 g.mol ⁻¹ | | | Arsenic | |
| Periodic table | Electronegativity accord | ling to Pâuling | 1.5 | | | Astatine | |
| Calculators | - seed a - 0 € - 0 100 300 1 | g to . daming | | | | Gold | |
| isitors information | Density | | 1.86 g.cm ⁻³ | | | Boron | |
| Our partners | Melting point | | 1280 °C | | | Barium | |
| contact us | Boiling point | | 2970 °C | | | Beryllium | |
| Security of the security of th | Vanderwaals radius | | unknown | (EEE) | | Bohrium | |
| Request a | Ionic radius | | unknown | | | Bismuth | |
| Quote ? | Isotopes | | 1 | | | Berkelium | |
| | b. N | | | * | | Bromine | |
| SHARE DEM_ | Electronic shell | | 1s ² 2s ² or [He] 2s ² | | | Carbon | 2 |
| | Energy of first ionisation | n | 899.2 kJ.mol ⁻¹ | | | Calcium | |
| | Energy of second ionisa | tion | 1757 kJ.mol ⁻¹ | | | Cadmium | |
| Launtach DV | Standard potential | | - 1.70 V | | | Cerium | |
| Lenntech BV | • | | Abbé René-Just Hauy in | | | Californium | |
| Rotterdamseweg 402 M 2629 HH Delft The Netherlands | Discovered by | | 1798 | | 1 | Curium | |
| tel: +31 15 261 09 00 | | | | | | Cobalt | |
| fax: +31 15 261 62 89 | Beryllium | | e | IR: | | Chromium | |
| e-mail: info@lenntech.com | alloys. Beryllium has one of | of the highest meltin | y, strong, light-weight, primarily g points of the light metals. It has | s excellent thermal conductivity | | Cesium | |
| 9 | resist oxidation when expo | | ed nitric acid and at standard tem | perature and pressures berylliu | m | Copper | |
| | Applications | | | | | Dubnium | |
| | Beryllium is used as an allo | ying agent in the p | roduction of beryllium-copper. The | anks to their electrical and | | Darmstadtium | |
| | thermal conductivity, high over a wide temperature re | strenght and hardne ange beryllium-cop | ess, non magnetic properties, goo per alloys are used in many applic | od resistance, dimensional stabil cations. A typical application of | ity | Dysprosium | |
| | beryllium-copper alloys is i Beryllium is also used in th | | erospace industries. ection diagnostic (it is transparent | to X-rays) and in the making of | F | Erbium | |
| | various computer equipme | nt. | | | | Einsteinium | |
| | Beryllium in the environme | ent | | | | Europium | |
| | | | m, in soil 6 ppm. Beryllium in soil in plants vary between 1 and 40 p | | on | Fluorine | |
| 5 × | which eat these plants. | | most important of which are ber | | d | Iron | |
| | phenacite. Precious forms | | | trandice, beryn, am ysoberyn, am | u | Fermium | |
| | Health effects of bery | yllium | | | | Francium | |
| | | | humans; in fact it is one of the m | | | Gallium | 2 8 |
| | pneumonia. | y narmful when hun | nans breathe it in, because it can | damage the lungs and cause | | Gadolinium | |
| | | | n is called berylliosis, a dangerous | | | Germanium | |
| | Breathing in beryllium in t | he workplace is who | he heart. In about 20% of all case at causes berylliosis. People that l | | ns | Hydrogen | |
| | are most susceptible to th | | th neonle that are hyperconcitive | to this chemical. These reaction | ne. | Helium | |
| | can be very heavy and the | ey can even cause a | th people that are hypersensitive person to be seriously ill, a cond tiredness and breathing problem | lition known as Chronic Berylliur | m | Hafnium | |
| | | and blueness of ha | nds and feet. Sometimes people | | | Mercury | |
| | | | n can also increase the chances of | f cancer development and DAIA | | Holmium | |
| | damage. | und Coo, berymun | . con also increase the chances of | cancer development and DNA | | Hassium | |
| | Emulacana - t-1 - ee . | 6 h 111 | | | | Iodine | |
| | Environmental effect | [2] | | - | | Indium | |
| | in the environment in sma | | esult of natural processes and hun and beryllium through production | | | Iridium | |
| | and oil. | | | | | Potassium | |

Industrial emissions will add beryllium to air and wastewater disposals will add beryllium to water. It usually settles in sediment. Beryllium as a chemical element occurs naturally in soils in small amounts, but human activities have also increased these beryllium levels. Beryllium is not likely to move deeper into the soil and groundwater.

In water, chemicals will react with beryllium, causing it to become insoluble. This is a good thing, because the water-insoluble form of beryllium can cause much less harm to organisms than the water-soluble form. Beryllium will not be accumulated in the bodies of fish. However, some fruits and vegetables such as kidney beans and pears may contain significant levels of beryllium. These levels can enter animals that eat them, but luckily most animals excrete beryllium quickly through urine and feces.

The uptake of beryllium has consequences mainly for human health. However, laboratory tests have indicated that it is possible for beryllium to cause cancer and changes of DNA with animals. So far there is no field evidence to support these findings.

Back to the periodic table of elements.

Lung Cancer Information View survivor stories & chat online w/ oncology information experts. CancerCenter.com/CareThe

Asbestos Lawsuits National Claims Assistance. Free Online Case Evaluation. Learn More Asbestos Action.org

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Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium Manganese

Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium

Oxygen Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Polonium

Praseodymium

Platinum Plutonium

Radium

Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Antimony

Scandium Selenium

Seaborgium

Silicon

Samarium

Tin

Strontium

Terbium

Technetium

Tellurium



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BERYLLIUM COMPOUNDS

CASRN: NO CAS RN

This record contains general information for beryllium ions and compounds, including statements in the literature referenced to beryllium compounds, beryllium salts, etc. For compound-specific information, refer to the appropriate individual records as listed in the RELATED HSDB RECORDS field; for information on the metal itself, refer to the BERYLLIUM, ELEMENTAL record.

For more information, search the NLM HSDB database.

Human Health Effects:

Toxicity Summary:

Exposure of people to relatively high concentrations of beryllium (greater than 100 ug cu m) causes acute beryllium disease, characterized by chemical pneumonitis. ... Some people inhaling low concentrations of beryllium develop chronic beryllium disease, a granulomatous lung disease characterized by dyspnea, cough, reduced pulmonary function, and a variety of other symptoms, including weight loss. ... The lack of a dose-response relationship between the extent of exposure and development of the disease, long latency period between exposure and onset, and the low incidence among beryllium-exposed individuals suggests that the disease is immune mediated. ... Occupational risk associated with exposure to beryllium-containing alloys has been documented for individuals exposed to berylliumcopper and beryllium-nickel alloys. Beryllium is a suspected human carcinogen, based on results of animal data. Epidemiologic evidence relating beryllium exposure to cancer in humans is inadequate to demonstrate or refute that beryllium is carcinogenic in humans, and the International Agency for Research on Cancer lists the evidence for beryllium induced carcinogenicity in humans as "limited". ... The pulmonary effects of inhaled beryllium have also been evaluated in a variety of laboratory animal species. ... monkeys exposed to relatively high concentrations of beryllium compounds developed symptoms and histopathological findings consistent with acute beryllium disease. ... Granulomatous lung disease has also been produced in guinea pigs exposed to beryllium compounds by inhalation or by intratracheal instillation. ... Repeated inhalation of beryllium-containing materials, including soluble beryllium compounds by various strains of laboratory rats has resulted in development of inflammatory and proliferative changes, granulomatous lung changes, and the development of lung tumors. Although beryllium- exposed rats have developed various degrees of granulomatous lung disease, none have developed immunopathological responses in lung or beryllium hypersensitivity. ... studies have shown that several strains of mice inhaling beryllium develop pulmonary lesions with features consistent with chronic beryllium disease. Lung lesions consisted of infiltration of lymphocytes into the lung interstitium, development of microgranulomas consisting of T lymphocytes and macrophages, and the presence of some pulmonary fibrosis. Under certain exposure conditions, increased numbers of lymphocytes were recovered in bronchoalveolar lavage fluid from exposed animals.

[Chang, L.W. (ed.). Toxicology of Metals. Boca Raton, FL: Lewis Publishers, 1996, p. 929-30] **PEER REVIEWED**

Evidence for Carcinogenicity:

Evaluation: There is sufficient evidence in humans for the carcinogenicity of beryllium and beryllium compounds. There is sufficient evidence in experimental animals for the carcinogenicity of beryllium and beryllium compounds. Overall evaluation: Beryllium and beryllium compounds are carcinogenic to humans (Group 1). /Beryllium and beryllium compounds/

[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization,
International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). Available at: http://monographs.iarc.fr/index.php
p. 58 103 (1993)] **PEER REVIEWED**

A1; Confirmed human carcinogen. /Beryllium & compounds, as Be/

[American Conference of Governmental Industrial Hygienists TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, 2008, p. 14] **QC REVIEWED**

WEIGHT OF EVIDENCE CHARACTERIZATION: B1; probable human carcinogen. Based on the limited evidence of carcinogenicity in humans exposed to airborne beryllium (lung cancer) and sufficient evidence of carcinogenicity in animals (lung cancer in rats and monkeys inhaling beryllium, lung tumors in rats exposed to beryllium via intratracheal instillation, and osteosarcomas in rabbits and possibly mice receiving intravenous or intramedullary injection), beryllium is reclassified from a B2 (inadequate human data) to a B1 probable human carcinogen (limited human data) using criteria of the 1986 Guidelines for Carcinogen Risk Assessment. Using the proposed Guidelines for

Animal Toxicity Studies:

Toxicity Summary:

Exposure of people to relatively high concentrations of beryllium (greater than 100 ug cu m) causes acute beryllium disease, characterized by chemical pneumonitis. ... Some people inhaling low concentrations of beryllium develop chronic beryllium disease, a granulomatous lung disease characterized by dyspnea, cough, reduced pulmonary function, and a variety of other symptoms, including weight loss. ... The lack of a dose-response relationship between

the extent of exposure and development of the disease. long latency period between exposure and onset, and the low incidence among beryllium-exposed individuals suggests that the disease is immune mediated. ... Occupational risk associated with exposure to beryllium-containing alloys has been documented for individuals exposed to berylliumcopper and beryllium-nickel alloys. Beryllium is a suspected human carcinogen, based on results of animal data. Epidemiologic evidence relating beryllium exposure to cancer in humans is inadequate to demonstrate or refute that beryllium is carcinogenic in humans, and the International Agency for Research on Cancer lists the evidence for beryllium induced carcinogenicity in humans as "limited". ... The pulmonary effects of inhaled beryllium have also been evaluated in a variety of laboratory animal species. ... monkeys exposed to relatively high concentrations of beryllium compounds developed symptoms and histopathological findings consistent with acute beryllium disease. ... Granulomatous lung disease has also been produced in guinea pigs exposed to beryllium compounds by inhalation or by intratracheal instillation. ... Repeated inhalation of beryllium-containing materials, including soluble beryllium compounds by various strains of laboratory rats has resulted in development of inflammatory and proliferative changes, granulomatous lung changes, and the development of lung tumors. Although beryllium- exposed rats have developed various degrees of granulomatous lung disease, none have developed immunopathological responses in lung or beryllium hypersensitivity. ... studies have shown that several strains of mice inhaling beryllium develop pulmonary lesions with features consistent with chronic beryllium disease. Lung lesions consisted of infiltration of lymphocytes into the lung interstitium, development of microgranulomas consisting of T lymphocytes and macrophages, and the presence of some pulmonary fibrosis. Under certain exposure conditions, increased numbers of lymphocytes were recovered in bronchoalveolar lavage fluid from exposed animals.

[Chang, L.W. (ed.). Toxicology of Metals. Boca Raton, FL: Lewis Publishers, 1996, p. 929-30] **PEER REVIEWED**

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| out Lenntech | Home > Periodic table > Elements > Boron | | | | Elements |
| rnkey plants | Boron - B | | | | Actinium |
| ot plants | | | | | Silver |
| ntainerized plants | Chemical properties of boron | - Health effects of boron - | Environmental effects of | f boron | Aluminum |
| rvices | Atomic number | 5 | | | Americium |
| reer at Lenntech | Atomic mass | 10.81 g.mol ⁻¹ | | | Argon |
| ternational Internships | Floring and late according to Bouling | | | | Arsenic |
| riodic table | Electronegativity according to Pauling | 2.0 | | | Astatine |
| Iculators | Density | 2.3 g.cm ⁻³ at 20°C | , a X | | Gold |
| sitors information | Melting point | 2076 °C | | | Boron |
| ır partners | Boiling point | 3927 °C | 1 14 | | Barium |
| ontact us | Vanderwaals radius | 0.098 nm | | | Beryllium |
| | | 0.027 nm | | | Bohrium |
| Request a | Ionic radius | | | 1. | Bismuth |
| Quote ? | Isotopes | 2 | 1 4 | 3470 | Berkelium |
| | Electronic shell | [He] 2s ² 2p ¹ | | | Bromine |
| | Energy of first ionisation | 800.5 kJ.mol ⁻¹ | | | Carbon |
| SHARE E E | Forms of account to stock to | 2.00 5.12 | | | Calcium |
| | Energy of second ionisation | 2426.5 kJ.mol ⁻¹ | | | Cadmium |
| | Energy of third ionisation | 3658.7 kJ.mol ⁻¹ | | | Cerium |
| Lenntech BV | Discovered by | Sir Humphry Davy and J.L | | | Californium |
| Rotterdamseweg 402 M | , | Gay-Lussac in 1808 | | | Chlorine |
| 2629 HH Delft The Netherlands | Boron | | | | Curium |
| tel: +31 15 261 09 00 | Boron is a non metallic element and the only | non-motal of the group 12 of the p | ariadis table the elements | | Cobalt |
| fax: +31 15 261 62 89 | Boron is electron-deficient, possessing a vaca | ant p-orbital. It has several forms, t | the most common of which is | | Chromium |
| e-mail: info@lenntech.com | amorphous boron, a dark powder, unreactive form borides. | | | | Cesium |
| | At standard temperatures boron is a poor ele | ectrical conductor but is a good cond | ductor at high temperatures. | | Copper |
| | Applications | | | | Dubnium |
| | The most economically important compound borax, used for insulating fiberglass and sodion | | | | Darmstadtium |
| | in textile products. Compounds of boron are used in organic synt | thesis, in the manufacture of a part | icular type of glasses, and as | | Dysprosium |
| | wood preservatives. Boron filaments are used and light weight. | | | | Erbium |
| | An early use of borax was to make perborate Boron compound also came into the average | | | | Einsteinium |
| | and fish. | , | | | Europium |
| | Boron in the environment | | | | Fluorine |
| | Boron is not present in nature in elemental for | | boric acid, kernite, ulexite, | | Iron |
| | colemanite and borates. Vulcanic spring wate Borates are mined in US, Tibet, Chile and Tur | | bout 2 million tonnes per | | Fermium |
| | year. | | | | Francium |
| | Health effects of boron | | | | Gallium |
| | Humans can be exposed to boron through fr a regular daily intake of about 2 mg and abo | | consumer products. we have | | Gadolinium |
| | When humans consume large amounts of bo | | centrations in their hodies | | Germanium |
| | may rise to levels that can cause health prol | blems. Boron can infect the stomac | h, liver, kidneys and brains | | Hydrogen |
| | and can eventually lead to death. When even | | | | Helium |
| | and can eventually lead to death. When exp nose, throat or eyes may occur. It takes 5 g its life in danger. | | | | |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. | | haven does not | | Harnium |
| | nose, throat or eyes may occur. It takes 5 g | | boron does not accumulate | | Hafnium |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. Eating fish or meat will not increase the bord within the tissues of animals. | | boron does not accumulate | | Mercury |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. Eating fish or meat will not increase the bord | | boron does not accumulate | | Mercury Holmium |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. Eating fish or meat will not increase the bord within the tissues of animals. | on concentrations in our bodies, as | | | Mercury Holmium Hassium |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. Eating fish or meat will not increase the borwithin the tissues of animals. Environmental effects of boron Boron is an element that occurs in the environment did | on concentrations in our bodies, as concentrations in our bodies. | cesses. | | Mercury Holmium Hassium Iodine |
| | nose, throat or eyes may occur. It takes 5 g its life in danger. Eating fish or meat will not increase the bord within the tissues of animals. Environmental effects of boron Boron is an element that occurs in the environmental effects of the env | on concentrations in our bodies, as on ment mainly through natural product to the release into air, soil and we amounts. Humans add boron by ment the addition of agricultural fertilize. | cesses. Water through weathering. It canufacturing glass, ers. The concentrations of | | Mercury Holmium Hassium |

dust in the workplace does exist. Boron exposure may also occur from consumer products such as cosmetics and laundry products.

Plants absorb boron from the ground and through plant-consuming animals it can end up in food chains. Boron has been found in animal tissue, but it is not likely to accumulate.

When animals absorb large amounts of boron over a relatively long period of time through food or <u>drinking water</u> the male reproductive organs will be affected. When animals are exposed to boron during pregnancy their offspring may suffer from birth defects or delayed development. Furthermore, animals are likely to suffer from nose irritation when they breathe in boron.

Now visit our boron in water page

Back to chart periodic elements

Recommended daily intake of boron



Krypton

Lanthanum

Lithium

Lawrencium

Lutetium Mendelevium

Magnesium

Manganese

Molybdenum

Meitnerium

Nitrogen

Sodium Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium

Oxygen Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium

Rubidium

Rhenium Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur Antimony

Scandium

Selenium

. . .

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium

Thorium

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| nkey plants | Cobalt - Co | | | Actinium | |
| ot plants | | | | Silver | |
| ntainerized plants | Chemical properties of cobal | t - Health effects of cobalt | - Environmental effects | of cobalt Aluminum | |
| rvices | Atomic number | 27 | | Americium | |
| reer at Lenntech | Atomic mass | 58.9332 g.mol ⁻¹ | | Argon | |
| ternational Internships | | | | Arsenic | |
| eriodic table | Electronegativity according to Pauling | 1.8 | | Astatine | |
| lculators | Density | 8.9 g.cm ⁻³ at 20°C | | Gold | |
| sitors information | Melting point | 1495 °C | | Boron | |
| ır partners | Boiling point | 2927 °C | r | Barium | |
| ontact us | Vanderwaals radius | 0.125 nm | AND DESCRIPTION OF | Beryllium | |
| | 1 | | | Bohrium | |
| Request a | Ionic radius | 0.078 nm (+2); 0.063 nm (+3) | Carlo par | Bismuth | |
| Quote ? | Isotopes | 8 | A 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 | Berkelium | |
| Secretary Secretary Secretary | Electronic shell | [Ar] 3d ⁷ 4s ² | | Bromine | |
| | Erectionic such | | | Carbon | |
| SHARE E L M. | Energy of first ionisation | 757 kJ.mol ⁻¹ | | Calcium | |
| | Energy of second ionisation | 1666.3 kJ.mol ⁻¹ | | Cadmium | |
| | Energy of third ionisation | 3226 kJ.mol ⁻¹ | | Cerium | |
| Lenntech BV | | 200 | | Californium | |
| Rotterdamseweg 402 M | Standard potential | - 0.28 V (Co ²⁺ / Co) ; +1.84 V (Co ³⁺ / Co ²⁺) | | Chlorine | |
| 2629 HH Delft The Netherlands | Discovered by | George Brandt in 1737 | | Curium | |
| tel: +31 15 261 09 00 | Discovered by | George Brandt III 1737 | | Cobalt | |
| fax: +31 15 261 62 89 | | | | Chromium | |
| e-mail: info@lenntech.com | Cobalt | | | Cesium | |
| | Cobalt is a hard ferromagnetic, silver-white periodic table. Like iron, it can be magnetize | | | | |
| | is active chemically, forming many compour attacked by dilute acids. | | | Dubnium | |
| | NO. 100 7007 | | | Darmstadtium | |
| | Applications | | | Dysprosium | |
| | Cobalt is used in many alloys (superalloys for high-speed steels, cemented carbides), in n | nagents and magnetic recording me | dia, as catalysts for the petro | | |
| | and chemical industries, as drying agents for and is used bu craft workers in porcelain, po | Einsteinium | | | |
| | isotopes, cobalt-60, is used in medical treat protect the consumer. | ment and also to irradiate food, in o | order to preserve the food and | Europium | |
| | Cobalt in the environment | | | Fluorine | |
| | Most of the Earth's cobalt is in its core. Coba | alt is of relatively low abundance in | the Earth's crust and in natura | Iron | |
| | waters, from which it is precipitated as the Although the average level of cobalt in soils | highly insoluble cobalt sulfine CoS. | | Farming | |
| | much as 70 ppm. In the marine environment nitrogen fixing organisms. Cobalt is not four | nt cobalt is needed by blue-green al | gae (cyanobacteria) and other | | |
| | usually not mined alone, and tends to be prores of cobalt are cobaltite, erythrite, glauc | oduced as a by-product of nickel an | d copper mining activities. The | e main | |
| | Democratic Republic of the Congo, mainland Azerbaijan, and Kazakhstan. | | | Gadolinium | |
| | World production is 17.000 tonnes per year. | | | Germanium | |
| | Health effects of cobalt | | | Hydrogen | |
| | As cobalt is widely dispersed in the environment and eating food that contains cobalt. Skin co | | | er Helium | |
| | exposure. Cobalt is not often freely available in the er | | The Settlement of the Settleme | iment Hafnium | |
| | particles the uptake by plants and animals | | | Mercury | |
| | Cobalt is beneficial for humans because it is | | | lt is Holmium | |
| | used to treat anaemia with pregnant wome daily intake of cobalt is variable and may be | | | al Hassium | |
| | unadsorbed, except that in vitamine B ₁₂ . | and the second producting to the commence of the commence | | Iodine | |
| | However, too high concentrations of cobalt concentrations of cobalt through air we exp | | | | |
| | occurs with people that work with cobalt. | | | Tridium | |
| | When plants grow on contaminated soils the of the plant we eat, such as fruits and seed: | | | parts Potassium | |

- Vomiting and nausea Vision problems
- Heart problems
- Thyroid damage

Health effects may also be caused by radiation of radioactive cobalt isotopes. This can cause sterility, hair loss, vomiting, bleeding, diarrhoea, coma and even death. This radiation is sometimes used with cancer-patients to destroy tumors. These patients also suffer from hair loss, diarrhea and vomiting.

Cobalt dust may cause an asthma-like disease with symptoms ranging from cough, shortness of breath and dyspnea to decreased pulmonary function, nodular fibrosis, permanent disability, and death. Exposure to colmay cause weight loss, dermatitis, and respiratory hypersensitivity. LD 50 (oral, rat)- 6171 mg/kg. (LD50 = Lethal dose 50 = Single dose of a substance that causes the death of 50% of an animal population from exposure to the substance by any route other than inhalation. LD50 is usually expressed as milligrams or grams of material per kilogram of animal weight (mg/kg or g/kg).)

Carcinogenicity- International Agency for Research on Cancer (IARC) haslisted cobalt and cobalt compounds within group 2B (agents which are possibly carcinogenic to humans). ACGIH has placed cobalt and inorganic compounds in category A3 (Experimental animal carcinogen- the agent is carcinogenic in experimental animals at a relatively high dose, by route(s), histologic type(s), or by mechanism(s) that are not considered relevant to worker exposure.) Cobalt has been classified to be carcinogenic to experimental animals by the Federal Republic of

Environmental effects of cobalt

Cobalt is an element that occurs naturally in the environment in air, water, soil, rocks, plants and animals. It may also enter air and water and settle on land through wind-blown dust and enter surface water through run-off when rainwater runs through soil and rock containing cobalt.

Humans add cobalt by releasing small amounts into the atmosphere from coal combustion and mining, processing

of cobalt-containing ores and the production and use of cobalt chemicals.

The radioactive isotopes of cobalt are not present in the environment naturally, but they are released through nuclear power plant operations and nuclear accidents. Because they have relatively short half-lives they are not

Cobalt cannot be destroyed once it has entered the environment. It may react with other particles or adsorb on soil particles or water sediments. Cobalt will only mobilize under acidic conditions, but ultimately most cobalt will end up in soils and sediments.

Soils that contain very low amounts of cobalt may grow plants that have a deficiency of cobalt. When animals graze on these grounds they suffer from lack of cobalt, which is essential for them.

On the other hand, soils near mining and melting facilities may contain very high amounts of cobalt, so that the uptake by animals through eating plants can cause health effects. Cobalt will accumulate in plants and in the bodies of animals that eat these plants, but cobalt is not known to bio magnify up the food chain. Because of this fruits, vegetables, fish and other animals we eat will usually not contain very high amounts of cobalt.

Back to chart periodic elements

Refractory Metals Tantalum, Tungsten, Molybdenum, Niobium. Extensive Inventory. www.cageallovs.com Need a Water Treatment? Kansas City's Best Water Treatment Never Buy Before Checking with Us! www.KcKinetico.com <u>High Conductivity</u> Nano- and Sub-Nanometer Silver Dispersions for Conductive Inks <u>www.nanogap-usa.com</u> AdChoices ▶

Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium

Manganese Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Nickel

Nobelium

Neptunium

Oxygen

Osmium

Phosphorus

Protactinium

Lead Palladium

Promethium

Praseodymium

Platinum

Plutonium

Radium Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Scandium

Selenium

Seaborgium

Silicon

Tin

Strontium

Terbium Technetium

Tellurium

Thorium

Cobalt Facts Environment

9 Cobalt in the Environment

The pages that follow give an overview of the role of cobalt in health and the environment.

For further information, please refer to the HS&E section on this website.



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Cobalt Exposure and Heart Disease (April 2006)

Cobalt in the form of Vitamin B_{12} (hydroxocyanocobalamin) is essential for humans. Vitamin B_{12} supports important synthetic reactions in metabolic processes and is essential for the production of red blood cells. The metabolism of Vitamin B_{12} and the daily ingestion of cobalt—containing foodstuffs provide the most significant source of cobalt (e.g. background level) in the human body. Background levels of cobalt are not known to be associated with adverse health effects in humans.

Humans may ingest up to several milligrams of cobalt per day in their diet and based on case reports, appear to tolerate even higher daily doses of cobalt during clinical treatment for anaemia without adverse effects to the heart. However, the ingestion of relatively high levels of cobalt (when compared to dietary intake) from inorganic cobalt salts with large amounts of alcohol has been reported to pose health risks to some humans. In the mid 1960's, small amounts of cobalt (1-2 parts per million) in the form of cobalt chloride were added to a brand of beer as a foam stabiliser. A number of fatalities related to cardiomyopathy were reported in men who consumed large amounts (more than 8 pints per day) of the cobalt-laden beer. Daily oral doses of cobalt in this group were in the range of 0.1 milligram per kilogram body weight.

Cardiomyopathy is a type of heart disease characterised by damage to the muscle and structure of the heart. The resultant effect is muscle cell death and a decrease in the volume of blood pumped. Studies of animal and human exposure to cobalt indicate that oral cobalt exposure, poor diet, and alcohol consumption are jointly associated with heart damage similar to that reported in beer drinkers.

A recent (2004) cross-sectional occupational exposure study of about 200 cobalt refinery workers found no clinically significant heart disease. However, among the most highly exposed workers, there was a relationship between cumulative cobalt exposure and alterations in left ventricular filling and relaxation times. The clinical significance of these changes is currently being investigated.

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Cobalt Exposure and Red Blood Cells (April 2006)

Cobalt is known to stimulate the production of red blood cells. A cobalt-iron medication was once used to treat specific types of anaemia. In clinical situations where decreased numbers of red blood cells were associated with anaemia, ingestion of inorganic cobalt (cobalt +2 ion in the form of cobalt chloride) stimulated an increase in the production of red blood cells (polycythemia or erythrocytosis). Typical adult doses were administered daily in the range of 50-100 milligrams of cobalt (0.7-2.0 milligrams cobalt per kilogram body weight).

Clinical treatments for sickle cell anaemia in children, utilizing higher cobalt doses (in the range of 3 to 4 milligrams per kilogram body weight), were associated with thyroid effects (decreased iodine uptake, e.g. goiter) in addition to the desired polycythemia. The thyroid and polycythemic effects were reversible upon cessation of oral cobalt treatment. Doses lower than 3 milligrams per kilogram body weight were not reported to induce thyroid effects in children. Currently, the clinical use of cobalt for the treatment of anaemia has been replaced by the use of synthetic erythropoetin, a hormone that induces the production of red blood cells.

Environmental cobalt exposures have been associated with altitude-induced polycythemia and Mountain Sickness* in some residents of a Peruvian mining village located 4300 meters above sea-level. High altitudes may also induce polycythemia in humans due to decreased oxygen levels. One study group of villagers, none of whom currently worked in the mine, with polycythemia (induced by high altitude) and significantly increased levels of cobalt measured in their blood (>1 microgram per litre), had greater frequency of Mountain Sickness when compared to villagers with polycythemia and normal cobalt blood levels.

Two occupational exposure studies have assessed the presence of polycythemia in cobalt-exposed workers. One study was conducted in a cobalt refinery and the other was conducted in a factory where cobalt dyes were used to paint porcelain plates. Both studies reported slight but non-significant decreases in red blood cells of workers. The prevalence and clinical significance of polycythemia in occupational cobalt exposures is not known.

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*Mountain Sickness: Polycythemia (erythrocytosis) usually develops at high altitudes due to lowered ambient oxygen levels. If the polycythemia is severe enough headache, dizziness, weakness, mental confusion, shortness of breath, decreased oxygen saturation, and death may occur. For review see:

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The Effect of Inhalation of Cobalt Substances on the Lungs. (April 2006)

Occupational inhalation exposures to elemental cobalt powders, salts, oxides and mixed-metal cobalt compounds such as hardmetal (cobalt-tungsten carbide) powders have been associated with asthma in workers. In some instances, antibodies to cobalt were detected in their blood. Workers exposed to cobalt-containing diamond dust in diamond polishing operations (which use cobalt powders as adhesives on the polishing disk) have been reported to undergo pathological changes in their lung tissue that range from intense inflammation to cellular infiltration and fibrosis. Some hardmetal workers have also been reported to contract this condition which has been named "hardmetal lung disease" or "hardmetal pneumoconiosis". Outside of diamond polishing and hardmetal operations, hardmetal disease has not been reported in workers exposed to cobalt substances alone. The prevalence of hardmetal lung disease in hardmetal operations is not known.

With respect to cobalt exposure (in the absence of mixed metals) in humans and lung cancer, there is one epidemiology study and a follow-up study investigating deaths in cobalt salt workers. The initial study reported four deaths due to lung cancer which would have been significantly higher than the expected deaths. However, the study authors later reclassified one lung cancer death after review of death certificates. Based on three deaths, the initial study results were not statistically significant, and the follow-up study did not report any increased deaths due to lung cancer.

There are four epidemiological evaluations of occupational hard metal exposure and lung cancer. While all four studies reported low but significantly increased deaths due to lung cancer, each study has confounding issues. These issues include: the effects of tobacco smoking and other life-style factors, incomplete current exposure data on workers, and lack of information on previous exposure history of the workers.

Animal studies include a lifetime (2-year) inhalation study of cobalt sulphate heptahydrate aerosol in rodents where lifetime inhalation exposure was associated with lung cancer (significant increases in bronchio-alveolar tumours) in rats and mice. Severe inflammation of the entire respiratory tract at all doses was reported as well. Studies with rodents and miniature swine have reported inflammation, lung fibrosis and emphysema after long-term inhalation or intra-tracheal instillation of cobalt, cobalt oxide and cobalt-tungsten carbide powders. Studies investigating the long-term inhalation of hardmetal powder and lung cancer in experimental animals have not been found in the scientific literature.

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The Effects of Cobalt Exposure on the Mammalian Reproductive System (November 2006)

Exposure to cobalt can occur through inhalation, oral or dermal (skin) routes. Mammals, including humans, are exposed to natural sources of cobalt in their food, water and air. In addition to naturally occurring forms in the environment, cobalt substances may also be present in certain occupational settings and in some consumer products.

Cobalt in the chemically distinct form of Vitamin B_{12} is essential for humans. While humans require Vitamin B_{12} , mammals such as deer, moose and elk, as well as domestic farm ruminants such as cattle and sheep, directly require the bioavailable cobalt (II) ion for reproductive health. It is common veterinary and agricultural practice to provide cobalt salt supplements to ensure a sufficient source of bioavailable cobalt (II) ion for animal health. Agricultural and veterinary experiences with bioavailable cobalt indicate there are "safe" doses which can maintain beneficial levels of cobalt in the animal. Doses of cobalt in diets that are too low (deficient) or too high (over-exposure) have been reported to have harmful effects. Cobalt-deficient diets are associated with a "wasting disease" in farm ruminants, deer, elk and moose where the animals fail to thrive and their reproductive output is significantly decreased. Over-exposures are associated with decreased reproductive output in farm ruminants.

Over-exposure to water-soluble cobalt salts (and thus the cobalt II ion) has been shown to cause damage to testicular tissue in male rats and mice. The effects are reported by oral exposure to cobalt chloride and by inhalation exposure to cobalt sulphate and they appear to be dose-dependent. These studies also reported sperm number and motility were affected. The oral and inhalation studies reported a threshold level (exposure dose) below which no adverse effects were observed for testicular tissue, sperm motility and sperm number. At the highest oral doses studied, fertility in male mice was significantly reduced (measured as the percentage of fertilised ova). A threshold level was also found for the adverse effects on fertility in male mice. It is unclear whether fertility in male mice is restored after the cessation of oral exposure.

Similar reproductive studies with female mice as well as other rodents and animals have not been found in the literature. In addition, no studies have been found with respect to the impact on the human reproductive system of exposure to elemental cobalt or cobalt substances

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Cobalt in Food (February 2006)

This is a short article on the occurrence of cobalt in food. It aims to present the variety of cobalt concentrations in different food groups.

Why is Cobalt found in food?

Cobalt (Co) is a natural element that is essential for the healthy functioning of many plants and animals and it is often found in the food that we eat.

What foods in Cobalt found in?

Research suggests that the top three food groups for Co in the human diet are: milk and dairy products, which account for approximately 32% of the total Co intake; fish and crustaceans, which account for approximately 20%, and condiments, sugar and oils, which account for about 16%. One investigation of specific foods (Leblanc *et al.*, 2004) found that chocolate contains the highest level of Co, with molluscs and crustaceans, and dried fruit and nuts also containing the high levels in comparison to other foods. Other studies show different Co levels for the same products in neighbouring countries (e.g. coffee levels as reported by Ostapczuk *et al.* 1987), and even within the same food type in a single country (Nigerian tea as reported by Onianwa et al. 1999; Chinese tea as reported by Qiu-e et al. 1999). For example, an American study found less than 0.05 mg/L of Co in white bread, pancakes, cheddar cheese and spaghetti with meatballs (Dolan and Capar, 1999). The Agency for Toxic Substances and Disease Registry (ATSDR) website contains a profile for Co, including its presence in a number of different foodstuffs, and references to various studies (e.g., 20 brands of beer as reported by Camean et al. 1998).

How much Cobalt is in an average diet?

One study used the figures in the accompanying Table and calculated Co levels in a typical diet (Leblanc *et al.* 2004). It was found that adults (over 15 years old) ingested around 7.5 μ g of Co per day (μ g = microgram = 1 x 10⁻⁶ grams) and children (aged 3 to 14 years) ingested approximately 7.3 μ g of Co per day. Another study found that the average Canadian male (40-65 years) had a 'mean daily dietary intake' of 12 μ g of Co per day, and that women of the same age group averaged 9 μ g of Co per day, with the differing levels mainly due to differences in diet between the sexes. (Dabeka and McKenzie 1995)

| Food Group | Concentration (mg/kg) |
|-------------------|-----------------------|
| Bread | 0.006 |
| Breakfast Cereals | 0.008 |
| Rice | 0.010 |

| Other Cereals | 0.001 |
|---------------------------------|-------|
| Milk | 0.001 |
| Cheese | 0.018 |
| Eggs and derivatives | 0.005 |
| Butter | 0.018 |
| Oils | 0.018 |
| Meats | 0.008 |
| Poultry and game | 0.002 |
| Offal ' | 0.033 |
| Fish | 0.007 |
| Crustaceans and molluscs | 0.046 |
| Vegetables (excluding potatoes) | 0.006 |
| Pulses | 0.008 |
| Fruits | 0.009 |
| Dried fruits and nuts | 0.041 |
| Chocolate | 0.050 |
| Sugar and derivatives | 0.021 |
| Soups | 0.006 |
| Ready meals | 0.008 |

Cobalt contents in various foodstuffs (Le Blanc et al, 2004)

What is Vitamin B12?

Cobalt in the form of vitamin B12 (cyanocobalamin) is essential for human health. The molecular formula for Vitamin B12 is $C_{63}H_{88}CoN_{14}O_{14}P$ which means that only 4.34% of the molecule by weight is cobalt. It is a coenzyme in a number of cellular processes including the oxidation of fatty acids and the synthesis of DNA. It also works with folic acid in the synthesis of certain amino acids, and is required for the normal production of red blood cells. Vitamin B12 is also essential for the nervous system.

International organizations responsible for setting standard levels of vitamin intake, the FAO (Food and Agricultural Organisation of the United Nations) and WHO (World Health Organisation) recommend a $2.4\mu g/day$ of vitamin B12 (equivalent to $0.1~\mu g/day$ of cobalt) in the adult diet.

(ftp://ftp.fao.org/es/esn/nutrition/Vitrni/pdf/TOTAL.pdf)

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ATSDR ToxFAQ's for Cobalt: http://www.atsdr.cdc.gov/tfacts33.html

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Cobalt in the Atmosphere (October 2006)

This short article aims to bring together information on Cobalt in the atmosphere.

What are the sources for Cobalt in the Air?

As Cobalt is a non-volatile material, it is rarely found in the atmosphere alone. Typically it is found in the air attached to anthropogenic pollutant particles, with a fraction of the cobalt found in natural dust. There are a number of anthropogenic sources for such particles, and natural sources of dust include the weathering of local geology, volcanic eruptions, forest fires and seawater spray. Coarse particles with diameters greater than $2\mu m$ may be deposited within 10km of the point of emission, whilst smaller particles may travel further. The mass median diameter of atmospheric cobalt has been found to be $2.6~\mu m$ (Milford and Davidson, 1985)

The main sources of atmospheric pollution for cobalt are industrial plants such as incinerators and chemical plants. In many countries, there is a limit on the amount of cobalt which such plants can release. Smaller sources for cobalt entering the atmosphere are exhaust fumes, the burning of fossil fuels (Vouk and Piver, 1983) and agriculture.

How much Cobalt is in the Atmosphere?

Friberg et al (1986) reported average natural background levels of cobalt in the atmosphere at around $1.0 \times 10^{-9} \text{mg/m}^3$ and stated that the atmospheric concentrations of cobalt in remote areas is very low (less than $1.0 \times 10^{-10} \text{mg/m}^3$ in the Antarctic) while in urban areas the ambient air concentration is usually higher (in the order of $1.0 \times 10^{-6} \text{ mg/m}^3$ and exceeding $1.0 \times 10^{-5} \text{mg/m}^3$ in heavily industrialised cities). Seiler et al. (1988) reported the cobalt concentrations in ambient air in several places in North and South America and in the United Kingdom and found that the levels were in the range 7.0×10^{-8} to $5.0 \times 10^{-6} \text{ mg/m}^3$. Over the open ocean, cobalt concentrations ranged from 0.0004 to 0.08 ng/m³ (Chester et al. 1991)

Khan et al (2003) investigating dustfall in Peshawar (Pakistan) found levels of cobalt at around 68µg per g of dustfall. In the paper these levels were ascribed to industrial uses such as the manufacture of alloys and its use as a catalyst in industry, although no direct evidence was given for this relationship.

Where does the Cobalt end up?

The length of time that cobalt stays in the atmosphere depends upon factors such as meteorological conditions, particle size, density and form. When the dust settles out of the atmosphere it can either land on soil, where it will eventually add to the soil concentration of cobalt, or into water, both of which are discussed in other documents available on this website.

Rainwater washes out any soluble cobalt species which are in the atmosphere. Studies have identified mean cobalt concentrations in rainwater to be between $0.3 \mu g/l$ in rural areas and $1.7 \mu g/l$ in highly industrial areas (Arimoto, 1985; Hansson et al, 1988). Recent data from the Swedish Environmental

Research Institute (ECOLAS, pers comm) indicate that cobalt levels in precipitation over rural areas were below 0.039 μ g/l between 2001 and 2003. Equally low values were seen in the Netherlands for 1999 and 2000 with the annual concentration of cobalt in rainwater being 0.001 μ mol/l, translating to a mean annual cobalt wet deposition rate of 4.1μ g/m² in the Netherlands for 1999 and 2000. In comparison a wet deposition level of 12μ g/m² was found in Massachusetts Bay as part of a total (wet and dry) of 47μ g/m² between September 1992 and September 1993 (Golomb *et al.* 1997).

Studies in the UK have shown that between 33 and 44% of the cobalt occurred as stable organic complexes (Nimmo and Fones, 1997), meaning it was not bio-available to soil dwelling or aquatic organisms. Total Cobalt deposition flux at a site in the Rhone delta in southern France in 1988-1989 was 0.42±0.23kg/km² year with 0.15 kg//km² year in the form of wet deposition (Guieu et al. 1991)

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Cobalt in Soils (February 2006)

This short article aims to explain the importance of cobalt in soils, concentrating on European Soils for which more information is currently available.

How much Cobalt is in European Soils?

The average Cobalt (Co) concentration in European soils is between 1- 20 mg/kg of Co (i.e. 1-20 mg of Co per kg of soil) dry weight, although this can become much higher in areas which are geologically rich in Co such as North Wales. For example, Paveley (1998) found natural levels of Co at over 2,500 mg/kg dry weight in soil. The study noted that the area had a totally healthy eco-system which had adapted to these naturally high concentrations. Maps of total Co and other metals in European Soils can be found on the FOREGS website (http://www.gsf.fi/publ/foregsatlas/index.php).

The majority of Co in the soil is not bioavailable. Co forms stable carbonate and hydroxide minerals which cannot be absorbed by the animal or plant life (Perez-Espinosa et al, 2004). Consequently, a very large amount of Co would have to be introduced into a volume of soil before local wildlife could be adversely affected.

How does Cobalt get into the Soil?

Cobalt occurs naturally in soils through two major pathways: the breakdown of organic matter which contains Vitamin B12, and the weathering of the local geology into soil particles. Mankind also adds Co to the soil, primarily through three mechanisms. The major mechanism is use of Co salts, e.g. Cobalt sulphate, as a feed additive to keep cattle and crops healthy in areas where there is not enough natural bioavailable Co. Smaller amounts of Co also enter the soil from the air transport of particulate emissions and application of sewage sludge onto fields.

Why is Cobalt added to some soils?

Due to the problems associated with Cobalt deficiency in agricultural soils, the behaviour of Co entering, and within, soils has been studied for a number of years. A lack of Co in a form which plants or earth dwelling organisms are able to absorb can have major effects on the health of the wildlife in an area. A classic example of this is the "Nova Scotia Moose Mystery" (Frank et al, 2004) where moose in Eastern North America were observed to have a wasting debilitating disease. It was found to be related to inadequate levels of bioavailable Co in their diet. The authors concluded

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that Co salt licks should be introduced in limited areas of Nova Scotia to balance the moose's diet and restore them to health.

Bioavailable Co in soil is also necessary for healthy functioning of some plants. This is especially true for leguminous plants, Co being an essential nutrient for the microorganisms which fix atmospheric nitrogen in the plants root nodules (Gad, 2002).

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DISCLAIMER

This summary is intended to provide general information about the topic under consideration. It does not constitute a complete or comprehensive analysis, and reflects the state of knowledge and information at the time of its preparation. This summary should not be relied upon to treat or address health, environmental, or other conditions.



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Cobalt in the Aquatic Environment (February 2006)

For the purposes of this short article, we have taken the aquatic environment to include freshwater systems such as lakes, streams and rivers and salt water or marine systems including estuaries and oceans.

What level of Cobalt is found in Natural Waters?

As Cobalt (Co) is naturally occurring and widely dispersed element, all natural waters contain a trace level of this element; sometimes called "background", i.e. the level that is present without any anthropogenic influence. This "background" level varies widely around the world and can even vary within the same water body. This can be due to water quality characteristics such as acidity or alkalinity (pH), temperature, and salinity, as well as, the plants and animals present and the composition of the sediments. For example, if Manganese is present in the sediment, then it will attract and adsorb almost all of the Co out of the water column and onto the mineral grains within the sediment. As a result, "nodules" form naturally in some areas of the ocean. One day Co may be recovered or extracted from these sediments as an alternative to terrestrial mining (i.e. mining on land).

The levels of Co found in surface waters of the Pacific and Atlantic Oceans are very low, averaging below 30 micrograms of Co per litre of sea water (i.e. 0.00003 g per litre). It should also be remembered that Co in the form of vitamin B12 is regarded as essential to the microbial ecology of the ocean.

In European freshwaters, the amount of dissolved Co is typically between 0.18-0.21 micrograms. Total concentrations vary between 0.22-0.5 micrograms, with the difference between total and dissolved being the proportion associated with organic matter in the water, e.g. humic acids. At these concentrations no toxicity to local aquatic organisms is expected to occur.

How does Co enter the aquatic environment?

Co can enter the aquatic environment from a number of sources, both natural and anthropogenic. The natural sources include volcanic emissions, the weathering of rocks by the action of water and decomposition of plant waste. One of the main human related releases into the aquatic environment is from sewage which, due to advances in modern sewage treatment, may only release around one part per billion (i.e. one unit Co for every billion units of sewage released) into the environment.

Where does Co go after the aquatic environment?

Natural and anthropogenic Co have the same transport pathways within the aquatic environment. The principle pathway involves binding to sediment at the bottom of the water body. Depending on the depositional environment, Co may remain on the sea/lake floor, become buried under further deposition or be transported into ocean where it will be deposited as sediment. The Co can be bound to sediments permanently unless there is a major change in the chemistry of the overlying waters. This is a natural geological process which, millions of years ago, created some of the ore bodies that are mined today.

Some Co, however, will remain in the water column (i.e. the water above the sediment). Co, if it is in the form of vitamin B12, is utilised by fauna in the environment. If this low level of Co in water was unavailable, a number of the aquatic organisms in these waters would show signs of vitamin B12 deficiency such as not achieving optimal growth or development.

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summary should not be relied upon to treat or address health, environmental, or other conditions.

| May 29th 2006 | Cobalt | Cobalt Oxide | Cobalt Sulphide | Cobalt Chloride | Cobalt Sulphate | Cobalt Carbonate | Cobalt Nitrate | Cobalt Acetate |
|------------------------------|-------------------|-------------------|-------------------|--------------------|--------------------|---------------------|-------------------|-----------------------|
| | CAS: 7440-48-4 | CAS: 1307-96-6 | CAS: 1317-42-6 | CAS: 7646-79-9 | CAS: 10124-43-3 | CAS: 513-79-1 | CAS: 10141-05-6 | CAS: 71-48-7 |
| | | * | | AND | AND | | AND | AND |
| | | | | Cobalt Chloride | Cobalt Sulphate | | Cobalt Nitrate | Cobalt Acetate |
| | | | | 6H ₂ 0 | 7H ₂ 0 | | 6H ₂ 0 | 4H ₂ 0 |
| Endpoint | | | | - | - | | | |
| | is: | | | CAS: 7791-13-1 | CAS: 10026-24-1 | | CAS: 10026-22-9 | CAS: 6147-53-1 |
| | | | | (NO EINECS) | (NO EINECS) | | (NO EINECS) | (NO EINECS) |
| | EINECS: 231-158-0 | EINECS: 215-154-6 | EINECS: 215-273-3 | EINECS: 231-589-4 | EINECS: 233-334-2 | EINECS: 208-169-4 | EINECS: 233-402-1 | EINECS: 200-755-8 |
| Physical Properties | None | None, | None | None | None | None | None | None |
| Acute Oral | None | Xn; R22 | None | Xn; R22 | Xn; R22 | None | None | None |
| Acute Inhalation | None | None | None | None | None | None | None | None |
| Dermal Irritation | Xi, R42/43 | R43 | R43 | R42/43 | R42/43 | R42/43 | R42/43 | R42/43 |
| Eye irritation | None | None | None | None | None | None | None | None |
| Dermal Sensitization | Xi, R42/43 | R43 | R43 | R42/43 | R42/43 | R42/43 | R42/43 | R42/43 |
| Respiratory Sensitisation | Xi, R42/43 | None | None | R42/43 | R42/43 | R42/43 | R42/43 | R42/43 |
| Chronic Toxicity | None | None | None | None | None | None | None | None |
| Reproductive Toxicity | None | None | None | Cat 2; R60 | Cat 2; R60 | Cat 2; R60 | Cat 2; R60 | Cat 2; R60 |
| Mutagenicity | None | None | None | Cat 3; R68 | Cat 3; R68 | Cat 3; R68 | Cat 3; R68 | Cat 3; R68 |
| Carcinogenicity | None | None | None | Cat 2; R49 | Cat 2; R49 | Cat 2; R49 | Cat 2; R49 | Cat 2; R49 |
| Aquatic Environment | R53 | N; R50/53 | N; R50/53 | N; R50/53 | N; R50/53 | N; R50/53 | N; R50/53 | N; R50/53 |
| Indications of Danger | Xi | Xn, N | Xi, N | T, N | T, N, | T, N, | T, N, | T, N, |
| S-Phrases | 2, 22, 24, 37, 61 | 2, 24, 37, 60, 61 | 2, 24, 37, 60, 61 | 53, 45, 60, 61 | 53, 45, 60, 61 | 53, 45, 60, 61 | 53, 45, 60, 61 | 53, 45, 60, 61 |

NOTE: Those classifications shaded grey do not enter force until the 30th ATP.

Specific Concentration Limits:

| Cobalt Sulphide | | | | | |
|-------------------------|------------------|--|--|--|--|
| Concentration | Classification | | | | |
| C ≥ 2,5 % | Xi, N; R43-50/53 | | | | |
| 1 % ≤ C < 2,5 % | Xi, N; R43-51/53 | | | | |
| 0,25 % ≤ C < 1 % | N; R51/53 | | | | |
| 0,025 % ≤ C < 0,25 % | N; R52/53 | | | | |

| Cobalt Chloride and Sulphate | | | | | |
|------------------------------|------------------------------------|--|--|--|--|
| Concentration | Classification | | | | |
| C ≥ 25 % | T, N; R49-60-22- 42/43-68-50/53 | | | | |
| 2,5 ≤ C < 25 % | T, N; R49-60- 42/43-68-50/53 | | | | |
| 1 % ≤ C < 2,5 % | T, N; R49-60- 42/43-68-51/53 | | | | |
| 0,5 % ≤ C < 1 % | T, N; R49-60- 51/53 | | | | |
| 0,25 % ≤ C < 0,5 % | T, N; R49-51/53 | | | | |
| 0,025 % ≤ C < 0,25 % | T; R49-52/53 | | | | |
| 0,01 % ≤ C < 0,025 % | T; R49 | | | | |

R22 = Harmful if swallowed

R43 = May cause sensitization by skin contact

R42/43 = May cause sensitisation by inhalation and skin contact

R49 = May cause cancer by inhalation

R50/53 = Very toxic to aquatic organisms, may cause long term adverse effects in aquatic environment

R53 = May cause long term adverse effects in the aquatic environment

R60 = May impair fertility

R68 = Possible risk of irreversible effects

S2 = Keep out of the reach of children

S22 = Do not breathe dust

S24 = Avoid Contact with skin

S37 = Wear Suitable Gloves

S45 = In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible).

S53 = Avoid exposure – obtain special instructions before use.

S60 = This material and its container must be disposed of as hazardous waste.

S61 = Avoid release to the environment. Refer to special instructions/safety data sheets.

| Cobalt Nitrates, Carbonate and Acetates | | | | | |
|--|---------------------------------|--|--|--|--|
| Concentration | Classification | | | | |
| C ≥ 2.5% | T, N; R49-60- 42/43-68-50/53 | | | | |
| 1 % ≤ C < 2,5 % | T, N; R49-60- 42/43-68-51/53 | | | | |
| 0,5 % ≤ C < 1 % | T, N; R49-60- 51/53 | | | | |
| 0,25 % ≤ C < 0.5 % | T, N; R49-51/53 | | | | |
| 0,025 % ≤ C < 0,25 % . | T; R49-52/53 | | | | |
| 0,01 % ≤ C < 0,025 % | T; R49 | | | | |

Note E: Substances with specific effects on human health (see chapter 4 of Annex VI of Directive 6715481EEC) that are classified as carcinogenic, mutagenic, and/or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are also classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances, the risk phrases R2O, R21, R22, R23, R24, R25. R26. R27, R28, R39, R68 (harmful), R48 and R65 and all combinations of these risk phrases shall be preceded by the word 'Also'.

Xi = Irritating

Xn = Harmful

T = Toxic

N = Dangerous for the environment

Direct and indirect effects of iron on river ecosystems

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The direct and indirect effects of iron on the structure and function of lotic ecosystems are reviewed. In addition to the mining of Fe enriched ores, intensified forestry, peat production and agricultural draining have increased the load of iron in many river ecosystems. The effects of iron on aquatic animals and their habitats are mainly indirect, although the direct toxic effects of Fe2+ are also important in some lotic habitats receiving Fe-enriched effluents in cold seasons, in particular. Ferric hydroxide and Fe-humus precipitates, on both biological and other surfaces, indirectly effect lotic organisms by disturbing the normal metabolism and osmoregulation, and by changing the structure and quality of benthic habitats and food resources. The combined direct and indirect effects of iron contamination decrease the species diversity and abundance of periphyton, benthic invertebrates and fishes. Sorption and co-precipitation of metals by Fe-oxides decrease the bioavailability and toxicity of water-borne metals, but may increase the dietary supply of metals and lead to toxic effects along the food chain. At the cellular level, Fe functions both as a detoxification mechanism, as well as a cell degeneration agent by inducing the formation of hydroxyl free radicals. More research is needed on the factors that affect the environmental fate and ecotoxicological impacts of iron on river ecosystems. As the flux of iron is heavily affected by seasonally varying physical, chemical and biological processes, its impact on ecosystems should be studied on multiple spatial, and temporal scales and at different levels of biological organisation.

1. Introduction

Iron has an essential role as a constituent of enzymes, such as cytochromes and catalase, and of oxygen-transporting proteins, such as haemoglobin and myoglobin. In fresh waters, iron is also an important nutrient for algae and other organisms. Due to its high abundance within the earth's crust, iron is ubiquitous in all freshwater environments and often reaches significantly higher concentra-

tions in water and sediments than other trace metals (Livingstone 1963, Förstner & Wittmann 1979).

High iron concentrations in fresh waters have long been considered a problem. In domestic use, iron-enriched waters may induce rust formation on plumbing fixtures, the staining of laundry and a metallic taste in drinking water. Hence, much effort has been put into the retention of iron in drinking water (Theis & Singer 1974). The mining of ironrich ores has caused the degradation of many river

enriched food may reduce the uptake of Fe via the gut membranes by inducing formation of thick Fehydroxide encrustations on the gut walls (Gerhardt 1995). Gerhardt (1995) only found Fe(II) occurring internally, in the eyes and trachea of the mayfly Leptophlebia marginata, whereas both Fe(II) and Fe(III) were found externally, on the body, gill and gut surfaces. Gerhardt and Westermann (1995) found a significantly higher uptake of Fe in the Leptophlebia marginata nymphs of a humic river, compared withh a clearwater river. This was presumably due to the impact of humic material and microbial activity on the speciation of Fe.

Information on the toxicity of iron to aquatic animals is scarce and somewhat contradictory (Table 2). Further, most laboratory bioassays have not separated the impact of dissolved Fe(II) and Fe particles. Fe(II) is considered to be more toxic to aquatic animals than Fe(III) (Gerhardt 1992). In many toxicity tests, the acidification of the test solution via the formation of ferric oxyhydroxide, after the addition of iron as chloride or sulphide, may have also contributed to the observed toxic effects on aquatic organisms (Dave 1985). In general, the toxicity of iron seems to be highest in acid conditions where Fe(II) predominates (Gerhardt 1992).

Acutely toxic concentrations of iron in the laboratory exposures of invertebrates usually vary between 3 and 400 mg, even within the same species (Table 2). An exceptionally low 4 d LC₅₀ value of 0.32 mgFe \times l⁻¹ was reported for *Ephemerella subvaria* by Warnick and Bell (1969). The US EPA water quality criteria for freshwater aquatic life is 1.0 mgFe \times l⁻¹. This is principally derived from field observations of the adverse effects of iron. Obviously, factors affecting the concentration and speciation of iron greatly contribute to the variation in estimated iron toxicity (Dave 1984, Maltby et al. 1987).

Peuranen et al. (1994) observed damage in the gills of one-summer-old brown trout exposed to Fe(II) and Fe(III) at pH 5 and 6, with and without humic material. The nominal total iron concentration of the exposure water was $2 \text{ mg} \times 1^{-1}$. Staining of whole gill arches indicated that at least some of the Fe was bound to the gills even in the presence of humic material. Gill damage, consisting of fusion of the lamellae and hypertrophy of the epithelial

cells, induced reduced oxygen uptake and impairment of the ion regulation of the Fe-exposed brown trout. The gill damage was more adverse at pH 5 than at pH 6 (Peuranen et al. 1994). Although not clearly distinguished as such, the results of Peuranen et al. (1994) reveal the combined effects of Fe accumulated within, and precipitated on the gills. The indirect effects of Fe precipitates are reviewed in Chapter 4.

A potential mechanism of iron toxicity includes its role in DNA and membrane damage. Vertebrate studies have shown that high cellular concentrations of iron, ferrous iron in particular, may cause cell degeneration. The mechanism of this process is not fully understood, but it includes the iron-catalysed auto-oxidation of dopamine, which in turn generates hydroxyl-free radicals. Together with oxygen, these free radicals are highly reactive and may cause site-specific oxidative damage when produced in excess. Excessive production of free radicals in tissues may be caused by increased free iron concentrations following, for example, haemorrhage or disorder in the iron metabolism (e.g. Halliwell & Gutteridge 1984, Fornstedt et al. 1990, Stevens & Kalkwarf 1990, Obata et al. 1993). Iron-enriched food may also cause elevated Fe levels in tissues (Jones et al. 1981, Luoma 1983).

In most laboratory bioassays, the toxicity of iron to aquatic animals has been attributed to the motion inhibiting or smothering effects of Fe-hydroxide or Fe-humic precipitates on gills, eggs or other surfaces. As these kinds of toxic actions limit an individual's access to essential resources, such as oxygen or food, they are considered here as indirect effects.

4. Indirect effects of iron on aquatic animals and their resources

4.1. Iron as a physical stressor

Formation of iron precipitates on biological surfaces has frequently been reported to effect the survival, reproduction and behaviour of aquatic animals (e.g. Walter 1966, Smith et al. 1973, Smith & Sykora 1976, Amelung 1982, Gerhardt 1992). Von Lukowicz (1976) concluded that the toxic effect of iron on rainbow trout, at neutral pH, was due to the

| Home | Applications Processes Systems Proc | ducts Library | Languages |
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| rvices | Atomic number 26 | | Americium |
| reer at Lenntech | Atomic mass 55.85 g.mol ⁻¹ | | Argon |
| ternational Internships | Electronegativity according to | | Arsenic |
| riodic table | Pauling 1.8 | ¥1 | Astatine |
| Iculators | Density 7.8 g.cm ⁻³ at 20°C | | Gold |
| sitors information | Melting point 1536 °C | | Boron |
| r partners | | | Barium |
| ntact us | Boiling point 2861 °C | | Beryllium |
| | Vanderwaalsradius 0.126 nm | | Bohrium |
| Request a | Ionic radius 0.076 nm (+2); 0.064 nm (+3) | | Bismuth |
| Quote ? | Isotopes 8 | | Berkelium |
| | Electronic shell [Ar] 3d ⁶ 4s ² | 120 Ac 120 | Bromine |
| C cuops E b E | | | Carbon |
| SHARE ELB_ | Energy of first ionisation 761 kJ.mol ⁻¹ | | Calcium |
| | Energy of second ionisation 1556.5 kJ.mol ⁻¹ | | Cadmium |
| | Energy of third ionisation 2951 kJ.mol ⁻¹ | | Cerium |
| Lenntech BV | - o.44 V (Fe ²⁺ / Fe) ; 0.77 V (Fe ³⁺ / | | Californium |
| Rotterdamseweg 402 M 2629 HH Delft | Standard potential Fe ²⁺) | | Chlorine |
| The Netherlands | Discovered by The ancients | | Curium |
| tel: +31 15 261 09 00 | | | Cobalt |
| fax: +31 15 261 62 89 | | | Chromium |
| -mail: info@lenntech.com | Iron | | Cesium |
| | Iron is a lustrous, ductile, malleable, silver-gray metal (group VIII of the <u>periodic table</u>). It four distinct crystalline forms. Iron rusts in dump air, but not in dry air. It dissolves readily in | n dilute acids. Iron is | Copper |
| | chemically active and forms two major series of chemical compounds, the bivalent iron (II), compounds and the trivalent iron (III), or ferric, compounds. | or ferrous, | Dubnium |
| | Applications | | Darmstadtium |
| | Iron is the most used of all the metals, including 95 % of all the metal tonnage produced wo | orldwide. Thanks to | Dysprosium |
| | the combination of low cost and high strength it is indispensable. Its applications go from for cars, from scredrivers to washing machines, from cargo ships to paper staples. | | Erbium |
| | Steel is the best known alloy of iron, and some of the forms that iron takes include: pig iron, steel, wrought iron, alloy steels, iron oxides. | , cast iron, carbon | Einsteinium |
| | | | Europium |
| | Iron in the environment | | Fluorine |
| | Iron is believed to be the tenth most abundant element in the universe. Iron is also the mos 34.6%) element making up the Earth; the concentration of iron in the various layers of the E | Earth ranges from high | Iron |
| | at the inner core to about 5% in the outer crust. Most of this iron is found in various iron oxi- minerals hematite, magnetite, and taconite. The earth's core is believed to consist largely o | | Fermium |
| | alloy. Iron is essential to almost living things, from micro-organisms to humans. | | Francium |
| | World production of new iron is over 500 million tonnes a year, and recycled iron add other Economically workable reserves of iron ores exceed 100 billion tonnes. The main mining are | eas are China, Brazil, | Gallium |
| | Australia, Russia and Ukraine, with sizeable amounts mined in the USA, Canada, Venezuela, | Sweeden and India. | Gadolinium |
| 9 | Health effects of iron | | Germanium |
| | Iron can be found in meat, whole meal products, potatoes and vegetables. The human body animal products faster than iron in plant products. Iron is an essential part of hemoglobin; t | | Hydrogen |
| | agent of the blood that transports oxygen through our bodies. | | Helium |
| | Iron may cause conjunctivitis, choroiditis, and retinitis if it contacts and remains in the tissue of excessive concentrations of iron oxide fumes or dusts may result in development of a ben | | Hafnium |
| | called siderosis, which is observable as an x-ray change. No physical impairment of lung fun associated with siderosis. Inhalation of excessive concentrations of iron oxide may enhance | ction has been | Mercury |
| | development in workers exposed to pulmonary carcinogens. LD50 (oral, rat) = 30 gm/kg. (L | .D50: Lethal dose 50. | Holmium |
| | Single dose of a substance that causes the death of 50% of an animal population from expos by any route other than inhalation. Usually expressed as milligrams or grams of material peweight (mol/long) | | Hassium |
| | weight (mg/kg or g/kg).) | | Iodine |
| | A more common problem for humans is iron deficency, which leads to anaemia. A man need intake pf 7 mg of iron and a woman 11 mg; a normal diet will generally provided all that is it | | Indium |
| | | | Iridium |
| | Environmental effects of iron | | Potassium |

plants, air and water. It is strongly advised not to let the chemical enter into the environment because it persists in the environment.

Read more on iron in water

Back to the periodic table of elements.

Recommended daily intake of iron



Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium

Manganese

Molybdenum

Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium

Oxygen

Osmium Phosphorus

Protactinium

Lead

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Antimony

Scandium

Selenium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium

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| urnkey plants | Selenium - Se | | | | | Actinium | |
| lot plants | | | | | | Silver | |
| ontainerized plants | Chemical properties of s | elenium - | Health effects of seleniun | n - Environmental effec | ts of selenium | Aluminum | |
| rvices | Atomic number | | 34 | | | Americium | |
| reer at Lenntech | Atomic mass | | 78.96 g.mol ⁻¹ | | | Argon | |
| ternational Internships | Electronegativity according t | o Pauling | 2.4 | | | Arsenic | |
| riodic table | | o radiling | | | | Astatine | |
| Iculators | Density | | 4.79 g.cm ⁻³ at 20°C | | | Gold | |
| sitors information | Melting point | | 217 °C | | | Boron | |
| r partners | Boiling point | | 688 °C | Self-desired and annual | | Barium | |
| ntact us | Vanderwaals radius | | 0.14 nm | | | Beryllium | |
| | | | 0.198 nm (-2); 0.042 nm | | | Bohrium | |
| Request a | Ionic radius | | (+6) | | | Bismuth | |
| Quote ? | Isotopes | | 9 | 3,5 | | Berkelium | |
| | Electronic shell | | [Ar] 3d ¹⁰ 4s ² 4p ⁴ | | | Bromine | |
| SHARE ELE. | Energy of first ionisation | | 940,7 kJ.mol ⁻¹ | | | Carbon | |
| as online as a sa | | | * an * 1 * ventuezenzo. | | | Calcium | |
| | Energy of second ionisation | | 2045 kJ.mol ⁻¹ | | | Cadmium | |
| | Energy of third ionisation Standard potential | | 2973.7 kJ.mol ⁻¹ - 0.77 V | | | Cerium | |
| Lenntech BV | | | Jone Borrolius 1917 | | | Californium | |
| Rotterdamseweg 402 M 2629 HH Delft | Discovered by | | Jons Berzelius 1817 | | | Chlorine | |
| The Netherlands | * | | | | | Curium | |
| tel: +31 15 261 09 00 | Selenium | | | | | Cobalt | |
| fax: +31 15 261 62 89 | Selenium is a non metallic chem | | | | | Chromium | |
| e-mail: info@lenntech.com | and physical properties it resemb | us powder, a | | ray crystalline metallike form | called | Cesium | |
| | | | ter, but dissolves in concentrated | | ens. | Copper | |
| | Applications | | | | | Dubnium | |
| | Selenium has good photovoltaic | and photocon | ductive properties, and it is used | extensively in electronics, su | ch as | Darmstadtium | |
| | | | econd largest use of selenium is in ed colour to glasses and enamels. | | | Dysprosium | |
| | | | ood supplements. Selenium can a as artistic use is to intensify and e | | and | Erbium | |
| | white photographic images. Other | er uses of sele | enium are in metal alloys such as nt in DC current. Selenium is used | the lead plates used in storag | | Einsteinium | |
| | | | um compounds are added to anti | | | Europium | |
| | Selenium in the environment | | | | | Fluorine | |
| | Selenium is among the rarer ele- in the atmosphere as metyl deriv | | | | | Iron | |
| | known selenium-conaining miner | rals, some of | | selenium - but all are rare an | d | Fermium | |
| | are Canada, USA, Bolivia and Ru | issia. Global i | ndustrial production of selenium i | s around 1500 tonnes a year | | Francium | |
| | about 150 tonnes of selenium are | | | | | Gallium | |
| | Well fertilized agricultural soil ge | nerally has al | . It is released through both natu bout 400 mg/ton since the eleme | nt is naturally present in phos | phate | Gadolinium | |
| | fertilizers ans is often added as a destroyed, but selenium does ha | | nt. In its natural form as an elem- to change form. | ent selenium cannot be create | ed or | Germanium | |
| • | | | because selenium settles from air | | | Hydrogen | |
| | immobile. Selenium that is immo | obile and will | When selenium in soils does not re not dissolve in water is less of a | risk for organisms. The oxyge | | Helium | |
| | levels in the soil and the acidity of increased acidity of soils is usual | | increase mobile forms of seleniu uman activities, such as industria | | | Hafnium | |
| | | | of exposure to its compounds will | | | Mercury | |
| | | trations of wa | ter-soluble selenium, the season | of the year, organic matter of | ontent | Holmium | |
| | determine its mobility. | | | | | Hassium | |
| | Agriculture cannot only increase surface water, as selenium is bro | | content in soil; it can also increa- irrigation drainage water. | se selenium concentrations in | | Iodine | |
| | Health effects of selenium | | | | | Indium | |
| | Sirecto di deletituti | | | | | | |

cereals and meat. Humans need to absorb certain amounts of selenium daily, in order to maintain good health. Food usually contains enough selenium to prevent disease caused by shortages.

Selenium uptake through food may be higher than usual in many cases, because in the past many selenium-rich fertilizers have been applied on farmland.

People that live near hazardous waste-sites will experience a higher exposure through soil and air. Selenium from hazardous waste-sites and from farmland will end up in groundwater or surface water through irrigation. This phenomenon causes selenium to end up in local <u>drinking water</u>, so that exposure to selenium through water will be temporarily increased.

People that work in metal industries, selenium-recovery industries and paint industries also tend to experience a higher selenium exposure, mainly through breathing. Selenium is released to air through coal and oil comb

People that eat a lot of grains that grow near industrial sites may experience a higher exposure to selenium through food. Exposure to selenium through drinking water may be increased when selenium from hazardous waste disposals ends up in water wells.

Exposure to selenium through air only comes about in the workplace usually. It can cause dizziness, fatigue and irritations of the mucous membranes. When the exposure is extremely high, collection of fluid in the lungs and bronchitis may occur.

Selenium uptake through food is usually high enough to meet human needs; shortages rarely occur. When shortages occur people may experience heart and muscle problems.

When selenium uptake is too high health effects will be likely to come about. The seriousness of these effects depends upon the concentrations of selenium in the food and how often this food is eaten. The health effects of various forms of selenium can vary from brittle hair and deformed nails, to rashes, heat, swelling of the skin and severe pains. When selenium ends up in the eyes people experience burning, irritation and tearing.

Selenium poisoning may become so severe in some cases that it can even cause death.

Overexposure of selenium fumes may produce accumulation of fluid in the lungs, garlic breath, bronchitis, pneumonitis, bronchial asthma, nausea, chills, fever, headache, sore throat, shortness of breath, conjunctivitis, vomiting, abdominal pain, diarrhea and enlarged liver. Selenium is an eye and upper respiratory irritant and a sensitizer. Overexposure may result in red staining of the nails, teeth and hair. Selenium dioxide reacts with moisture to form selenious acid, which is corrosive to the skin and eyes. Carcinogenicity- The International Agency for Research on Cancer (IARC) has listed selenium within Group 3 (The agent is not classifiable as to its carcinogenicity to humans.)

Effects of selenium on the environment

Low levels of selenium can end up in soils or water through weathering of rocks. It will than be taken up by plants or end up in air when it is adsorbed on fine dust particles. Selenium is most likely to enter the air through coal and oil combustion, as selenium dioxide. This substance will be converted into selenium acid in water or sweat.

Selenium substances in air are usually broken down to selenium and $\underline{\text{water}}$ fairly quickly, so that they are not dangerous to the health of organisms.

The behaviour of selenium in the environment strongly depends upon its interactions with other compounds and the environmental conditions at a certain location at a certain time.

There is evidence selenium can accumulate in the body tissues of organisms and can than be passed up through the food chain. Usually this bio magnification of selenium starts when animals eat a lot of plants that have been absorbing large amounts of selenium, prior to digestion. Due to irrigation run-off concentrations of selenium tend to be very high in aquatic organisms in many areas.

When animals absorb or accumulate extremely high concentrations of selenium it can cause reproductive failure and birth defects.

Sources of periodic table.

Back to the periodic table of elements

Recommended daily intake of selenium



Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

ragnesium

Manganese Molybdenum

Meitnerium

Nitrogen

....

Sodium

Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium Oxygen

Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Polonium

Platinum

Plutonium

riucomui

Radium Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Antimony

Scandium Selenium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium

| Home | Applications Proc | esses | Systems | Products | Library | Languages |
|----------------------------------|--|------------------------------|--|---|-----------------|--------------|
| out Lenntech | Home > Periodic table > Elements > | · Vanadium | | | | Elements |
| rnkey plants | Vanadium - V | | | | | Actinium |
| ot plants | | | | | | Silver |
| ntainerized plants | Chemical properties of van | adium - <u>Health effe</u> | cts of vanadium | - Environmental effe | cts of vanadium | Aluminum |
| rvices | Atomic number | 23 | | | | Americium |
| reer at Lenntech | Atomic mass | 50.9414 g.r | mol ⁻¹ | | | Argon |
| ernational Internships | Electronegativity according to | Pauling 1.6 | | | | Arsenic |
| iodic table | | 2 | | | | Astatine |
| culators | Density | 6.1 g.cm ⁻³ | at 20°C | | | Gold |
| itors information | Melting point | 1910 °C | | | | Boron |
| r partners | Boiling point | 3407 °C | | | | Barium |
| ntact us | Vanderwaals radius | 0.134 nm | | 3 | <i>b</i> | Beryllium |
| | | 0.074 nm / | +3); 0.059 | 1000 | | Bohrium |
| Request a | Ionic radius | (+5) | +3); 0.039 | A . | E. W | Bismuth |
| Quote ? | Isotopes | 5 | | 100 | Value . | Berkelium |
| | Electronic shell | [Ar] 3d ³ 4 | .c2 | 1 | 100 | Bromine |
| | | | | | | Carbon |
| SHARE ELS. | Energy of first ionisation | 649.1 kJ.m | ol ⁻¹ | | | Calcium |
| | Energy of second ionisation | 1414 kJ.mo | ol ⁻¹ | | | Cadmium |
| | Energy of third ionisation | 2830 kJ.mo | _{sl} -1 | | | Cerium |
| Lenntech BV | 1 | | | | | Californium |
| Rotterdamseweg 402 M | Energy of fourth ionisation | 4652 kJ.mo | ol -1 | | | Chlorine |
| 2629 HH Delft The Netherlands | Discovered by | Nils Sefstro | m in 1830 | | | Curium |
| tel: +31 15 261 09 00 | | | | | | Cobalt |
| fax: +31 15 261 62 89 | Vanadium | | | | | Chromium |
| -mail: info@lenntech.com | Vanadium is a rare, soft, ductile gr | av-white element found c | ombined in certain r | ninerals and used mainly to | | Cesium |
| | produce certain alloys. Vanadium r oxidation states of vanadium include | esists corrosion due to a | | | | Copper |
| | Applications | | | | | Dubnium |
| | | | | -1-1101 | | Darmstadtium |
| | Most of the vanadium (about 80%) aluminium in titanium alloys is use | d in jet engines and high : | speed air-frames, ar | d steel alloys are used in ax | | Dysprosium |
| | crankshafts, gears and other critical vanadium has low neutron-adsorpt | | | | | Erbium |
| | Vanadium oxide (V ₂ O ₅) is used as | a catalyst in manufacturin | ng sulfuric acid and n | naleic anhydride and in maki | ng | Einsteinium |
| | ceramics. It is added to glass to pr infrared radiation at some specific | | Glass coated with va | nadium dioxide (VO ₂) can b | lock | Europium |
| | Vanadium in the environment | | | | | Fluorine |
| | | | | | | Iron |
| | Vanadium is never found unbound patronite, vanadinite, carnotite and | | | | | Fermium |
| | coal, oil shale and tar sands. Various vanadium ores are known | | | | ra iil al | Francium |
| | byproducts of other ores. The large production of vanadium ore is around the second sec | | | | | Gallium |
| | tonnes per year. Watering is an important way in w | nich vanadium is redistrib | uted around the env | ironment because venedates | are | Gadolinium |
| | generally very soluble. | | | | | Germanium |
| | Vanadium is abundant in most soils availability. | , in variable amounts, an | id it is taken up by p | lants at levels that reflect its | 8 | Hydrogen |
| | In biology, a vanadium atom is an | essential component of so | ome enzymes, partio | cularly the vanadium nitroge | nase | Helium |
| | used by some <u>nitrogen</u> -fixing micro | | TOTAL TOTAL CONTRACTOR OF THE PARTY OF THE P | | | Hafnium |
| | Health effects of vanadium | | | | | Mercury |
| | Vanadium compounds are not rega were found to suffer severe eye, n | | nowever, workers ex | posed to vanadium peroxide | dust | Holmium |
| | | | web feed-toff | as bushink | aliva | Hassium |
| | The uptake of vanadium by humar oil, sunflower oil, apples and eggs. | | ougn rooastuffs, such | as buckwheat, soya beans, | olive | Iodine |
| | Vanadium can have a number of e | | | o high. When vanadium upta | ake | Indium |
| | takes places through air it can cau | | | W | | Iridium |
| | The acute effects of vanadium are | irritation of lungs, throat, | eyes and nasal cav | ties. | | |

- Cardiac and vascular disease
- Inflammation of stomach and intestines
 Damage to the nervous system
- Bleeding of livers and kidneys Skin rashes
- Severe trembling and paralyses
- Nose bleeds and throat pains
 Weakening
- Sickness and headaches Dizziness
- Behavioural changes

The health hazards associated with exposure to vanadium are dependent on its oxidation state. This product contains elemental vanadium. Elemental vanadium could be oxidized to vanadium pentoxide during welding. The pentoxide form is more toxic than the elemental form. Chronic exposure to vanadium pentoxide dust and fumes may cause severe irritation of the eyes, skin, upper respiratory tract, persistent inflammations of the trachea and bronchi, pulmonary edema, and systemic poisoning. Signs and symptoms of overexposure include; conjunctivitis, nasopharyngitis, cough, labored breathing, rapid heart beat, lung changes, chronic bronchitis, skin pallor, greenish-black tongue and an allergic skin rash.

Effects of vanadium on the environment

Vanadium can be found in the environment in algae, plants, invertebrates, fishes and many other species. In mussels and crabs vanadium strongly bioaccumulates, which can lead to concentrations of about 10^5 to 10^6 times greater than the concentrations that are found in seawater.

Vanadium causes the inhibition of certain enzymes with animals, which has several neurological effects. Next to the neurological effects vanadium can cause breathing disorders, paralyses and negative effects on the liver and

Laboratory tests with test animals have shown, that vanadium can cause harm to the reproductive system of male animals, and that it accumulates in the female placenta.

Vanadium can cause DNA alteration in some cases, but it cannot cause cancer with animals.

Back to chart periodic elements

Recommended daily intake of vanadium

Welding Fume Extractor Portable Compact Hepa Filtered Unit Efficient, Effective Fume Control www.sentryair.com OSHA Safety Keep Your Employees Safe & Up To Date On OSHA Industry Standards! www.safetyservicescompany.com Free NFPA Diamond Guide Understand the NFPA Diamond at a glance. Use as a desk or wall chart www AdChoices D Krypton

Lanthanum

Lithium

Lawrencium

Lutetium

Mendelevium

Magnesium

Manganese

Molybdenum

Meitnerium Nitrogen

Sodium

Niobium

Neodymium

Neon

Nickel

Neptunium Oxygen

Osmium

Phosphorus

Protactinium

Palladium

Promethium

Polonium

Platinum Plutonium

Radium

Rubidium

Rutherfordium

Roentgenium

Rhodium

Radon

Sulfur

Antimony

Silicon

Samarium

Strontium

Tantalum

Terbium

Technetium

ATSDR Agency for Toxic Substances & Disease Registry ToxFAQs™ for for Antimony

(Antimonio (/es/toxfaqs/es_tfacts23.html))

September 1995

CAS#: 7440-36-0

[2] (/toxfaqs/tfacts23.pdf) PDF Version, 94 KB (/toxfaqs/tfacts23.pdf)

This fact sheet answers the most frequently asked health questions about antimony. For more information, you may call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

Summary

Exposure to antimony occurs in the workplace or from skin contact with soil at hazardous waste sites. Breathing high levels of antimony for a long time can irritate the eyes and lungs, and can cause problems with the lungs, heart, and stomach. This chemical has been found in at least 403 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is antimony?

Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide.

Little antimony is currently mined in the United States. It is brought into this country from other countries for processing. However, there are companies in the United States that produce antimony as a by-product of smelting lead and other metals.

Antimony isn't used alone because it breaks easily, but when mixed into alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics to prevent them from catching fire. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass.

What happens to antimony when it enters the environment?

- Antimony is released to the environment from natural sources and from industry.
- In the air, antimony is attached to very small particles that may stay in the air for many days.
- Most antimony ends up in soil, where it attaches strongly to particles that contain iron, manganese, or aluminum.
- Antimony is found at low levels in some rivers, lakes, and streams.

How might I be exposed to antimony?

- Because antimony is found naturally in the environment, the general population is exposed to low levels of it every day, primarily in food, drinking water, and air.
- It may be found in air near industries that process or release it, such as smelters, coal-fired plants, and refuse incinerators.
- In polluted areas containing high levels of antimony, it may be found in the air, water, and soil.
- Workers in industries that process it or use antimony ore may be exposed to higher levels.

How can antimony affect my health?

Exposure to antimony at high levels can result in a variety of adverse health effects.

Breathing high levels for a long time can irritate your eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers.

In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, problems with fertility have been seen when rats breathed very high levels of antimony for a few months.

Ingesting large doses of antimony can cause vomiting. We don't know what other effects may be caused by ingesting it. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it.

Antimony can have beneficial effects when used for medical reasons. It has been used as a medicine to treat people infected with parasites.

How likely is antimony to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified antimony as to its human carcinogenicity.

Lung cancer has been observed in some studies of rats that breathed high levels of antimony. No human studies are available. We don't know whether antimony will cause cancer in people.

Is there a medical test to show whether I've been exposed to antimony?

Tests are available to measure antimony levels in the body. Antimony can be measured in the urine, feces, and blood for several days after exposure. However, these tests cannot tell you how much antimony you have been exposed to or whether you will experience any health effects. Some tests are not usually performed in most doctors' offices and may require special equipment to conduct them.

Has the federal government made recommendations to protect human health?

The EPA allows 0.006 parts of antimony per million parts of drinking water (0.006 ppm). The EPA requires that discharges or spills into the environment of 5,000 pounds or more of antimony be reported.

The Occupational Safety and Health Administration (OSHA) has set an occupational exposure limit of 0.5 milligrams of antimony per cubic meter of air (0.5 mg/m³) for an 8-hour workday, 40-hour workweek.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) currently recommend the same guidelines for the workplace as OSHA.

Glossary

Carcinogenicity: Ability to cause cancer.

CAS: Chemical Abstracts Service.

Ingestion: Taking food or drink into your body.

Long-term: Lasting one year or more.

Milligram (mg): One thousandth of a gram.

Parasite: An organism living in or on another organism.

ppm: Parts per million.

Short-term: Lasting 14 days or less.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. <u>Toxicological Profile for antimony (/ToxProfiles/TP.asp?id=332&tid=58)</u>. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

If you have questions or concerns, please contact your community or state health or environmental quality department or:

For more information, contact:

Agency for Toxic Substances and Disease Registry Division of Toxicology and Environmental Medicine 1600 Clifton Road NE, Mailstop F-62

Atlanta, GA 30333

Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)

Fax: 1-770-488-4178

Email: cdcinfo@cdc.gov (mailto:cdcinfo@cdc.gov)

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Information line and technical assistance:

Phone: 888-422-8737 FAX: (770)-488-4178

To order toxicological profiles, contact:

National Technical Information Service 5285 Port Royal Road Springfield, VA 22161

Phone: 800-553-6847 or 703-605-6000

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- · Page last reviewed: March 3, 2011
- Page last updated: March 3, 2011
- Content source: Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/)

Agency for Toxic Substances and Disease Registry, 4770 Buford Hwy NE, Atlanta, GA 30341

Contact CDC: 800-232-4636 / TTY: 888-232-6348



| Home | Applications Processes | Systems Products | Library | Languages | | |
|----------------------------------|--|---|----------------|---------------------|--|--|
| bout Lenntech | Home > Periodic table > Elements > Antimony | | | Elements | | |
| urnkey plants | Antimony - Sb | | | Actinium | | |
| Pilot plants | • | | | Silver | | |
| Containerized plants | Chemical properties of antimony - I | Health effects of antimony - Environmental effec | ts of antimony | Aluminum | | |
| Services | Atomic number | 51 | | Americium | | |
| Career at Lenntech | Atomic mass | 121.75 g.mol ⁻¹ | | Argon | | |
| nternational Internships | | | | Arsenic | | |
| eriodic table | Electronegativity according to Pauling | 1.9 | | Astatine | | |
| alculators | Density | 6.684 g.cm ⁻³ | | Gold | | |
| isitors information | Melting point | 631 °C | | Boron | | |
| ur partners | Boiling point | 1380 °C | | Barium | | |
| ontact us | Vanderwaals radius | 0.159 nm | | Beryllium | | |
| | Validel Waais Tadius | 0.135 IIII | | Bohrium | | |
| Request a | Ionic radius | 0.245 nm (-3); 0.062 nm (+5); 0.076 nm (+3) | | Bismuth | | |
| Quote ? | Testones | | | Berkelium | | |
| | Isotopes | 12 | A PARSON | Bromine | | |
| | Electronic Shell | [Kr] 4d ¹⁰ 5s ² 5p ³ | | Carbon | | |
| SHARE DYM. | Energy of first ionisation | 834 kJ.mol ⁻¹ | | Calcium | | |
| | Energy of second ionisation | 1595 kJ.mol ⁻¹ | | Cadmium | | |
| | | | | Cerium | | |
| Lenntech BV | Energy of third ionisation | 2443 kJ.mol ⁻¹ | | Californium | | |
| Rotterdamseweg 402 M | Standard potential | 0.21 V (Sb ³⁺ / Sb) | | Chlorine | | |
| 2629 HH Delft The Netherlands | Discovered by | The ancients | | Curium | | |
| tel: +31 15 261 09 00 | | | | Cobalt | | |
| fax: +31 15 261 62 89 | Antimony | | | Chromium | | |
| e-mail: info@lenntech.com | | which can exist in two forms: the metallic form is bright, silvery | | Cesium | | |
| 41 | | ey powder. Antimony is a poor conductor of heat and electricity, acids or alkalis. Antimony and some of its alloys expand on cool | | Copper | | |
| | Antimony has been known since ancient times | . It is sometimes found free in nature, but is usually obtained fr | om | Dubnium | | |
| | | D ₃). Nicolas Lémery, a French chemist, was the first person to Is. He published his findings in 1707. Antimony makes up about | | Darmstadtium | | |
| | 0.00002% of the earth's crust. | | | Dysprosium | | |
| | Applications | | | Erbium | | |
| | Very pure antimony is used to make certain ty | Einsteinium | | | | |
| | detectors. Antimony is alloyed with <u>lead</u> to inc low friction metals, type metal and cable shea | Europium | | | | |
| | antimony, in the form of stibnite, for black eye | ic enamels, glass and pottery. The ancient Egyptians used make-up. | | Fluorine | | |
| | Antimony in the environment | | | Iron | | |
| | Antimony occurs naturally in the environment. | But it also enters the environment through several application | s by | Fermium | | |
| | | humans. Antimony is an important metal in the world economy. Annual production is about 50.000 tonnes per year, with virgin materials coming mainly from china, Russia, Bolivia and South Africa. World reserves exceed 5 | | | | |
| | million tonnes. In Finland there is a deposit of | | | Francium Gallium | | |
| | Health effects of antimony | | | Gadolinium | | |
| | | n suffer the effects of exposure by breathing in antimony dusts. by breathing air, drinking water and eating foods that contain it | | Germanium | | |
| | | ther substances that contain it. Breathing in antimony that is bo | | Hydrogen | | |
| | Exposure to relatively high concentrations of a | antimony (9 mg/m ³ of air) for a longer period of time can cause | | Helium | | |
| | | h effects may occur, such as lung diseases, heart problems, | | Hafnium | | |
| | diarrhea, severe vomiting and stomach ulcers It is not known whether antimony can cause of | ancer or reproductive failure. | | Mercury | | |
| | were sensitive to it have experienced health e | ections, but people who have had too much of the medicine or iffects in the past. These health effects have made us more awa | re | Holmium | | |
| | of the dangers of exposure to antimony. | | | | | |
| | Effects of antimony on the environm | nent | | Hassium | | |
| | Antimony can be found in soils, waters and air | in very small amounts. Antimony will mainly pollute soils. | | Iodine | | |
| | | nces towards other locations and surface waters. pigs have shown us that relatively high levels of antimony may | kill | Indium | | |
| | small animals. Rats may experience lung, hea | | | Iridium | | |

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Heat breathed in low levels of antimony for a crypte munths mall expense fertiliti publicus

Whether antimony can cause cancer has not been fully specified yet.

Sources of periodic table.

Back to the periodic table of elements.

Durammo Deep Anode System

Factory Assembled & Tested Anode Reliable, Durable, Longer Life www.matcor.com

Krypton

Lanthanum

Lithium

Lutetium

Mendelevium

Magnesium

Manganese

Molybdenum Meitnerium

Nitrogen

Sodium

Niobium

Neodymium

Nickel

Nobelium

Neptunium Oxygen

Osmium

Phosphorus

Protactinium

Lead

Palladium

Promethium

Praseodymium

Platinum Plutonium

Radium

Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Scandium Selenium

Seaborgium

Silicon

Tin

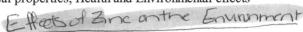
Strontium

Tantalum

Terbium

Technetium Tellurium

| Home | Applications Processe | s Systems | Products | Library | Languages |
|----------------------------------|---|--|--|------------------|-----------------|
| oout Lenntech | Home > Periodic table > Elements > Zinc | | | E | lements |
| nkey plants | Zinc - Zn | , | | A | ctinium |
| ot plants | | | | s | ilver |
| ntainerized plants | Chemical properties of | zinc - Health effects of zinc - | Environmental effects of | zinc A | luminum |
| vices | Atomic number | 30 | | A | mericium |
| eer at Lenntech | Atomic mass | 65.37 g.mol ⁻¹ | | A | rgon |
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| Rotterdamseweg 402 M | | | | | hlorine |
| 2629 HH Delft The Netherlands | | | | | urium |
| tel: +31 15 261 09 00 | Zinc | | | | obalt |
| fax: +31 15 261 62 89 | Zinc is a lustrous bluish-white metal. It is ordinary temperatures, but it becomes o | luctile and malleable when heated betw | een 110°C and 150°C. It is a fai | rly | hromium |
| -mail: info@lenntech.com | reactive metal that will combine with ox hydrogen. | ygen and other non-metals, and will re | act with dilute acids to release | | esium |
| | Applications | | | c | opper |
| | It is used principally for galvanizing iron, | | | | ubnium |
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| | single tablet, it is believed to possess an muscles of the body. | | insteinium | | |
| | Zinc in the environment | | | | uropium |
| | Zinc is a very common substance that or | ccurs naturally. Many foodstuffs contain | certain concentrations of zinc. D | rinking F | luorine |
| | water also contains certain amounts of z or toxic waste sites may cause the zinc a | | | | ron |
| | Zinc occurs naturally in air, water and so | il, but zinc concentrations are rising uni | naturally, due to addition of zinc | F | ermium |
| | through human activities. Most zinc is ac and steel processing. Some soils are hea | ded during industrial activities, such as wily contaminated with zinc, and these | mining, coal and waste combustare to be found in areas where | | rancium |
| | has to be mined or refined, or were sew | | | | allium |
| | Zinc is the 23rd most abundant element sphalerite. Other important zinc ores are | | | re G | adolinium |
| | Canada, Russia, Australia, USA and Peru exploitable reserves exceed 100 million | '. World production exceeds 7 million to | onnes a year and commercially | | ermanium |
| | Health effects of zinc | | | : - E | lydrogen |
| | Zinc is a trace element that is essential | for human health. When people absorb | too little zinc they can experience | te a | lelium |
| | loss of appetite, decreased sense of tast cause birth defects. | | | | afnium |
| | Although humans can handle proportion | ally large concentrations of zinc, too mu | uch zinc can still cause eminent h | ealth | lercury |
| | problems, such as stomach cramps, skir damage the pancreas and disturb the pr | irritations, vomiting, nausea and anae | mia. Very high levels of zinc can | H | olmium |
| | chloride can cause respiratory disorders | | | | lassium |
| | In the work place environment zinc cont pass after two days and is caused by over | | own as metal fever. This condition | on will I | odine |
| | Zinc can be a danger to unborn and new | born children. When their mothers hav | e absorbed large concentrations | of zinc | ndium |
| | the children may be exposed to it through | | | | ridium |
| | | | | P | otassium |



The world's zinc production is still rising. This basically means that more and more zinc ends up in the environment.

Water is polluted with zinc, due to the presence of large quantities of zinc in the wastewater of industrial plants. This wastewater is not purified satisfactory. One of the consequences is that rivers are depositing zinc-polluted sludge on their banks. Zinc may also increase the addity of waters.

Some fish can accumulate zinc in their bodies, when they live in zinc-contaminated waterways. When zinc enters the bodies of these fish it is able to bio magnify up the food chain.

Large quantities of zinc can be found in soils. When the soils of farmland are polluted with zinc, animals will absorb concentrations that are damaging to their health. Water-soluble zinc that is located in soils can contaminate groundwater.

Zinc cannot only be a threat to cattle, but also to plant species. Plants often have a zinc uptake that their systems cannot handle, due to the accumulation of zinc in soils.

On zinc-rich soils only a limited number of plants has a chance of survival. That is why there is not much plant diversity near zinc-disposing factories. Due to the effects upon plants zinc is a serious threat to the productions of farmlands. Despite of this zinc-containing manures are still applied.

Finally, zinc can interrupt the activity in soils, as it negatively influences the activity of microrganisms and earthworms. The breakdown of organic matter may seriously slow down because of this.

Read more on zinc in water

Back to the periodic table of elements.

Recommended daily intake of zinc

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Magnesium

Manganese

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maroge

Sodium

Niobium

Neodymium

Neon

Nickel

Nobelium

Neptunium

Oxygen

Osmium

Protactinium

Lead

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium Rubidium

Rhenium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Antimony

Scandium Selenium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium

ATSDR Agency for Toxic Substances & Disease Registry ToxFAQs™ for Zinc

(Cinc (/es/toxfaqs/es_tfacts6o.html))

August 2005

CAS#: 7440-66-6

[2] (/toxfaqs/tfacts60.pdf) PDF Version, 29 KB (/toxfaqs/tfacts60.pdf)

This fact sheet answers the most frequently asked health questions about zinc. For more information, you may call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

Highlights

Zinc is a naturally occurring element. Exposure to high levels of zinc occurs mostly from eating food, drinking water, or breathing workplace air that is contaminated. Low levels of zinc are essential for maintaining good health. Exposure to large amounts of zinc can be harmful. It can cause stomach cramps, anemia, and changes in cholesterol levels. Zinc has been found in at least 985 of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is zinc?

Zinc is one of the most common elements in the earth's crust. It is found in air, soil, and water, and is present in all foods. Pure zinc is a bluish-white shiny metal.

Zinc has many commercial uses as coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass, and bronze. A zinc and copper alloy is used to make pennies in the United States.

Zinc combines with other elements to form zinc compounds. Common zinc compounds found at hazardous waste sites include zinc chloride, zinc oxide, zinc sulfate, and zinc sulfide. Zinc compounds are widely used in industry to make paint, rubber, dyes, wood preservatives, and ointments.

What happens to zinc when it enters the environment?

- Some is released into the environment by natural processes, but most comes from human activities like mining, steel production, coal burning, and burning of waste.
- It attaches to soil, sediments, and dust particles in the air.
- Rain and snow remove zinc dust particles from the air.
- · Depending on the type of soil, some zinc compounds can move into the groundwater and

into lakes, streams, and rivers.

- Most of the zinc in soil stays bound to soil particles and does not dissolve in water.
- It builds up in fish and other organisms, but it does not build up in plants.

How might I be exposed to zinc?

- Ingesting small amounts present in your food and water.
- Drinking contaminated water or a beverage that has been stored in metal containers or flows through pipes that have been coated with zinc to resist rust.
- Eating too many dietary supplements that contain zinc.
- Working in any of the following jobs: construction, painting, automobile mechanics, mining, smelting, and welding; manufacture of brass, bronze, or other zinc-containing alloys; manufacture of galvanized metals; and manufacture of machine parts, rubber, paint, linoleum, oilcloths, batteries, some kind of glass, ceramics, and dyes.

How can zinc affect my health?

Zinc is an essential element in our diet. Too little zinc can cause problems, but too much zinc is also harmful.

Harmful effects generally begin at levels 10-15 times higher than the amount needed for good health. Large doses taken by mouth even for a short time can cause stomach cramps, nausea, and vomiting. Taken longer, it can cause anemia and decrease the levels of your good cholesterol. We do not know if high levels of zinc affect reproduction in humans. Rats that were fed large amounts of zinc became infertile.

Inhaling large amounts of zinc (as dusts or fumes) can cause a specific short-term disease called metal fume fever. We do not know the long-term effects of breathing high levels of zinc.

Putting low levels of zinc acetate and zinc chloride on the skin of rabbits, guinea pigs, and mice caused skin irritation. Skin irritation will probably occur in people.

How likely is zinc to cause cancer?

The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have not classified zinc for carcinogenicity. Based on incomplete information from human and animal studies, the EPA has determined that zinc is not classifiable as to its human carcinogenicity.

How can zinc affect children?

Zinc is essential for proper growth and development of young children. It is likely that children exposed to very high levels of zinc will have similar effects as adults. We do not know whether children are more susceptible to the effects of excessive intake of zinc than the adults.

We do not know if excess zinc can cause developmental effects in humans. Animal studies have found decreased weight in the offspring of animals that ingested very high amounts of zinc.

How can families reduce the risks of exposure to zinc?

• Children living near waste sites that contain zinc may be exposed to higher levels of zinc

- through breathing contaminated air, drinking contaminated drinking water, touching or eating contaminated soil.
- Discourage your children from eating soil or putting their hands in their mouths and teach them to wash their hands frequently and before eating.
- If you use medicines or vitamin supplements containing zinc, make sure you use them appropriately and keep them out of the reach of children.

Is there a medical test to show whether I've been exposed to zinc?

There are tests available to measure zinc in your blood, urine, hair, saliva, and feces. These tests are not usually done in the doctor's office because they require special equipment. High levels of zinc in the feces can mean high recent zinc exposure. High levels of zinc in the blood can mean high zinc consumption and/or high exposure. Tests to measure zinc in hair may provide information on long-term zinc exposure; however, the relationship between levels in your hair and the amount of zinc you were exposed to is not clear.

Has the federal government made recommendations to protect human health?

The EPA recommends that drinking water should contain no more than 5 milligrams per liter of water (5 mg/L) because of taste. The EPA requires that any release of 1,000 pounds (or in some cases 5,000 pounds) into the environment be reported to the agency.

To protect workers, the Occupational Safety and Health Administration (OSHA) has set an average limit of 1 mg/m³ for zinc chloride fumes and 5 mg/m³ for zinc oxide (dusts and fumes) in workplace air during an 8-hour workday, 40-hour workweek.

Similarly, the National Institute for Occupational Safety and Health (NIOSH) has set the same standards for up to a 10-hour workday over a 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. <u>Toxicological Profile for Zinc (/ToxProfiles/TP.asp?id=302&tid=54)</u> (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information?

If you have questions or concerns, please contact your community or state health or environmental quality department or:

For more information, contact:

Agency for Toxic Substances and Disease Registry Division of Toxicology and Environmental Medicine 1600 Clifton Road NE, Mailstop F-62 Atlanta, GA 30333

Atlanta, OA 30333

Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)

Fax: 1-770-488-4178

Email: cdcinfo@cdc.gov)

ATSDR can also tell you the location of occupational and environmental health clinics. These

clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Information line and technical assistance:

Phone: 888-422-8737 FAX: (770)-488-4178

To order toxicological profiles, contact:

National Technical Information Service 5285 Port Royal Road Springfield, VA 22161 Phone: 800-553-6847 or 703-605-6000

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- Page last reviewed: March 3, 2011
- Page last updated: March 3, 2011
- Content source: Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/)

Agency for Toxic Substances and Disease Registry, 4770 Buford Hwy NE, Atlanta, GA 30341

Contact CDC: 800-232-4636 / TTY: 888-232-6348





Zinc

Zinc is a metal that is normally found in small amounts in nature. It is used in many commercial industries and can be released into the environment during mining and smelting (metal processing) activities. People living near smelters or industries using zinc could be exposed to higher levels of zinc by drinking water, breathing air and touching soil that contains the metal. Exposure to high levels of zinc over long periods of time may cause adverse health effects.

How does zinc get into the environment?

Although zinc occurs naturally, most zinc finds its way into the environment because of human activities. Mining, smelting metals (like zinc, lead and cadmium) and steel production, as well as burning coal and certain wastes can release zinc into the environment. A common use for zinc is to coat steel and iron as well as other metals to prevent rust and corrosion; this process is called galvanization. High levels of zinc in soil may result from the improper disposal of zinc-containing wastes from metal manufacturing industries and electric utilities. In soil, most of the zinc stays bound to the solid particles. When high levels of zinc are present in soils, such as at a hazardous waste site, the metal can seep into the groundwater.

Industries also can release dust containing higher levels of zinc into the air we breathe. Eventually, the zinc dust will settle out onto the soil and surface waters. Rain and snow also can remove zinc dust from the air. Most of the zinc in lakes, rivers and streams does not dissolve, but settles to the bottom. Some fish in these waters may contain high levels of zinc. High levels of zinc in the soil, water and air are often found along with high levels of other metals like lead and cadmium.

How might I be exposed to zinc?

Zinc can enter the body if you eat foods or drink water or other beverages containing zinc, or if you breathe zinc dust or fumes from the air. Very small amounts of zinc enter the body through skin contact. People living near a smelter or another zinc-producing industry who have a private well are most likely to be exposed through their drinking water. They also may be exposed to zinc through the air they breathe, or through vegetables grown in contaminated soils. Through frequent hand-to-mouth contact, children also can be exposed to zinc by playing in contaminated soils.

Foods naturally contain zinc but vary greatly in their zinc content. Very small amounts of the zinc in food are absorbed by the body. Drinking beverages stored in metal cans or drinking water that flows through metal pipes coated with zinc also are sources of zinc exposure.

How can zinc affect my health?

Zinc is an essential nutrient needed by the body for growth, development of bones, metabolism and wound-healing. Too little zinc in the diet also can cause adverse health effects such as loss of appetite, decreased sense of taste and smell, lowered ability to fight off infections, slow growth, slow wound-healing and skin sores.

A short-term illness called *metal fume fever* can result if workers breathe very high levels of zinc dust or fumes. This condition, which usually lasts from 24 to 48 hours, causes chills, fever, excessive sweating and weakness. Long-term effects of breathing zinc dust or fumes are not known.

Eating or drinking too much zinc in a short period of time can lead to adverse health effects, such as stomach cramps, nausea and vomiting. Eating large amounts of zinc for longer periods may cause anemia, nervous system disorders, damage to the pancreas and lowered levels of "good" cholesterol. There is no evidence that zinc causes cancer in humans.

How can I reduce my exposure to zinc?

Knowing possible sources of zinc in your environment is important in reducing exposure. Children are particularly at risk if they play in contaminated soil. Limiting play in contaminated areas and planting grass to provide ground cover can greatly reduce exposure. Practice good hygiene habits by washing children's hands and faces often, especially before eating and bedtime. Practice good housekeeping techniques by removing shoes before entering your home to prevent tracking contaminated soils inside, and vacuum carpets, rugs and upholstery often.

If you live near a smelter or another zinc-producing industry and your drinking water comes from a private well, you may want to have the water tested for zinc. Public water systems are tested for zinc on a regular basis. If you live near a zinc smelter or another industry that produces zinc, you also may want to have your garden soil tested.

Is there a medical test that can tell me if I have been exposed to zinc?

Zinc can be measured in blood, feces, urine and hair. Different tests may be able to show the type of exposure to zinc. High levels of zinc in the blood or feces might show a recent high exposure. Zinc levels measured in hair would show long-term exposure. However, these tests are not routinely used. If you think you have been exposed to elevated levels of zinc, contact your doctor for more information.

Where can I get more information?

Illinois Department of Public Health Division of Environmental Health Toxicology Section 525 W. Jefferson St. Springfield, IL 62761 217-782-5830 TTY (hearing impaired use only) 800-547-0466

This fact sheet was supported in part by funds from the Comprehensive Environmental Response, Compensation, and Liability Act trust fund through a cooperative agreement with the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services.

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Questions or Comments

ATSDR Agency for Toxic Substances & Disease Registry $ToxFAQs^{\text{TM}}$ for Thallium

(Talio (/es/toxfaqs/es tfacts54.html) (/es/toxfaqs/es tfacts54.html))

September 1995

CAS#: 7440-28-0

(/toxfaqs/tfacts54.pdf) PDF Version, 174 KB (/toxfaqs/tfacts54.pdf)

This fact sheet answers the most frequently asked health questions about thallium. For more information, you may call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

Summary

Exposure to thallium occurs mainly from eating food. Exposure to higher levels of thallium may occur in the workplace. Breathing high levels of thallium may result in effects on the nervous system, while ingesting high levels of it results in vomiting, diarrhea, temporary hair loss, and other effects. This chemical has been found in at least 210 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is thallium?

Pure thallium is a bluish-white metal that is found in trace amounts in the earth's crust. In the past, thallium was obtained as a by-product from smelting other metals; however, it has not been produced in the United States since 1984. Currently, all the thallium is obtained from imports and from thallium reserves.

In its pure form, thallium is odorless and tasteless. It can also be found combined with other substances such as bromine, chlorine, fluorine, and iodine. When it's combined, it appears colorless-to-white or yellow.

Thallium is used mostly in manufacturing electronic devices, switches, and closures, primarily for the semiconductor industry. It also has limited use in the manufacture of special glass and for certain medical procedures.

What happens to thallium when they enters the environment?

- Thallium enters the environment primarily from coal-burning and smelting, in which it is a trace contaminant of the raw materials.
- It stays in the air, water, and soil for a long time and is not broken down.
- Some thallium compounds are removed from the atmosphere in rain and snow.
- It's absorbed by plants and enters the food chain.

• It builds up in fish and shellfish.

How might I be exposed to thallium?

- Eating food contaminated with thallium may be a major source of exposure for most people.
- Breathing workplace air in industries that use thallium.
- Smoking cigarettes.
- Living near hazardous waste sites containing thallium (may result in higher than normal exposures).
- Touching or, for children, eating soil contaminated with thallium.
- Breathing low levels in air and water.

How can thallium affect my health?

Exposure to high levels of thallium can result in harmful health effects. A study on workers exposed on the job over several years reported nervous system effects, such as numbness of fingers and toes, from breathing thallium.

Studies in people who ingested large amounts of thallium over a short time have reported vomiting, diarrhea, temporary hair loss, and effects on the nervous system, lungs, heart, liver, and kidneys. It has caused death. It is not known what the effects are from ingesting low levels of thallium over a long time.

Birth defects were not reported in the children of mothers exposed to low levels from eating vegetables and fruits contaminated with thallium. Studies in rats, however, exposed to high levels of thallium, showed adverse developmental effects.

It is not known if breathing or ingesting thallium affects human reproduction. Studies showed that rats that ingested thallium for several weeks had some adverse reproductive effects. Animal data suggest that the male reproductive system may be susceptible to damage by low levels of thallium.

There is no information available on the health effects of skin contact with thallium in people or animals.

How likely is thallium to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified thallium as to its human carcinogenicity.

No studies are available in people or animals on the carcinogenic effects of breathing, ingesting, or touching thallium.

Is there a medical test to show whether I've been exposed to thallium?

There are medical tests available to measure levels of thallium in urine and hair. In addition, thallium can also be measured in blood; however, this is not a good indicator of exposure since thallium only stays in blood a very short time.

These tests require special equipment that is not usually available in most doctor's offices. In addition, these tests cannot determine if adverse health effects will occur from the exposure to thallium.

Has the federal government made recommendations to protect human health?

The EPA requires that discharges or accidental spills into the environment of 1,000 pounds or more of thallium be reported.

The Occupational Safety and Health Administration (OSHA) has set an exposure limit of 0.1 milligrams per cubic meter (0.1 mg/m³) for thallium in workplace air. The American Conference of Governmental Industrial Hygienists (ACGIH) has established the same guidelines as OSHA for the workplace.

The National Institute for Occupational Safety and Health (NIOSH) has recommended that 15 mg/m³ of thallium be considered immediately dangerous to life and health. This is the exposure level of a chemical that is likely to cause permanent health problems or death.

Glossary

Carcinogenicity: Ability to cause cancer.

Ingesting: Taking food or drink into your body.

Milligram (mg): One thousandth of a gram.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for thallium (/ToxProfiles/TP.asp?id=309&tid=49). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

If you have questions or concerns, please contact your community or state health or environmental quality department or:

For more information, contact:

Agency for Toxic Substances and Disease Registry Division of Toxicology and Environmental Medicine 1600 Clifton Road NE, Mailstop F-62

Atlanta, GA 30333

Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)

Fax: 1-770-488-4178

Email: cdcinfo@cdc.gov (mailto:cdcinfo@cdc.gov)

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

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• Page last reviewed: March 3, 2011

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|----------------------------------|--|--|-----------------------------------|---------------|-------------|--|--|--|
| out Lenntech | <u>Home</u> > <u>Periodic table</u> > <u>Elements</u> > <i>Thalliur</i> | n | | | Elements | | | |
| rnkey plants | Thallium - Tl | | | | Actinium | | | |
| ot plants | | | | | Silver | | | |
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| vices | Atomic number | 81 | | | Americium | | | |
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| iodic table | Electronegativity according to Pauling | 1.8 | | | Astatine | | | |
| culators | Density | 11.71 g.cm ⁻³ at 20°C | | | Gold | | | |
| tors information | Melting point | 304 °C | A . | | Boron | | | |
| partners | Boiling point | 1473 °C | | 12 | Barium | | | |
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| | Energy of first ionisation | 589.1 kJ.mol ⁻¹ | | | Carbon | | | |
| SHARE SE | Energy of second ionisation | 1970.5 kJ.mol ⁻¹ | | | Calcium | | | |
| | Energy of second formsactors | 1970.5 KJ.MOI | | | Cadmium | | | |
| | Energy of third ionisation | 2877.4 kJ.mol ⁻¹ | | | Cerium | | | |
| Lenntech BV | Discovered by | Sir William Crookes in 1861 | | | Californium | | | |
| Rotterdamseweg 402 M | | | | | Chlorine | | | |
| 2629 HH Delft The Netherlands | Thallium | | | | Curium | | | |
| tel: +31 15 261 09 00 | When freshly exposed to air, thallium exhibits a metallic lustre, but soon develops a blueish-grey tinge, | | | | | | | |
| fax: +31 15 261 62 89 | resembling lead in appearance. A heavy oxide builds up on thallium if left in air, and in the presence of water the hydroxide is formed. The metal is very soft and malleable. It can be cut with a knife. Chromium | | | | | | | |
| -mail: info@lenntech.com | Applications | | | | Cesium | | | |
| | Thallium is used for making low-melting po | | | | Copper | | | |
| | reagents in chemical research. Thallium sul pesticide, although banned in Western coun | Dubnium | | | | | | |
| | | infrared light, it is used in photocells. It is used for sink-float separation of minerals. Thallium amalgam is used in thermometers for low temperature, because it freezes at -58 °C (pure mercury freezes at -38 °C). | | | | | | |
| | Thallium in the environment | | | | Dysprosium | | | |
| | Thallium is not a rare element; it is 10 time | s more abundant than silver. The | element is widely dispersed ma | inly | Erbium | | | |
| | in potassium minerals such as sylvite and p crookesirte and lorandite. World production | ollucite. Thallium minerals are rare | e, but a few are known, such as | | Einsteinium | | | |
| | no assessment of how great the reserves a | o tonnes per year. There has be | en | Europium | | | | |
| | Thallium is partially water-soluble and cons | | | | Fluorine | | | |
| | amounts of the component. Thallium can all is fairly mobile within soils. | so spread by adsorption on sludge | . There are indications that that | lium | Iron | | | |
| | Health effects of thallium | Fermium | | | | | | |
| | The element and its compounds are toxic a | nd should be handled carefully. | | | Francium | | | |
| | Thallium occurs in the environment natural | Gallium | | | | | | |
| | as rat poison and as a substance in electro- exposure to thallium substances. | Gadolinium | | | | | | |
| | The human body absorbs thallium very effe | Germanium | | | | | | |
| | digestive tract. | | | | Hydrogen | | | |
| | Thallium poisoning is mainly caused by acci sulphate. Consequently, stomachaches will | Helium | | | | | | |
| | damage is so irreversible that death will so consequences of disturbances of the nervou | Hafnium | | | | | | |
| | remain. With unborn children thallium poise | Mercury | | | | | | |
| * | Due to accumulation of thallium in the bodi depressions, lack of appetite, leg pains, ha | | | s, | Holmium | | | |
| | | | | nces | Hassium | | | |
| | of thallium uptake through food. | her effects that can be related to thallium poisoning are nerve pains and joint pains. These are consequences lallium uptake through food. | | | | | | |
| | Effects of thallium on the Environ | Iodine Indium | | | | | | |
| | | | | r E | | | | |
| | There has been no significant contamination its neighbors in the periodic table, mercury | | rom industry, unlike that caused | 1 Dy | Iridium | | | |

upon plants, such as colour changes in leaves and growth declines. Mammals, such as rabbits, are just as susceptible to the toxic effects of thallium as humans.

Back to periodic table elements.

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Krypton

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Mendelevium

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Manganese

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Meitnerium Nitrogen

Sodium

Niobium

Neodymium

Neon

Nickel

Nobellum

Neptunium

Oxygen

Osmium Phosphorus

Protactinium

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium Rubidium

Rutherfordium

Roentgenium

Rhodium

Radon

Ruthenium

Sulfur

Antimony Scandium

Selenium

Seaborgium

Silicon

Samarium

Tin

Strontium

Tantalum

Terbium

Technetium

Tellurium

Thorium



INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY

ENVIRONMENTAL HEALTH CRITERIA 182

THALLIUM

This report contains the collective views of an international group of experts and does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International Labour Organisation, or the World Health Organization.

First draft prepared by Professor G. Schaub, Institute of Zoology and Parasitology, Ruhr University, Bochum, Germany

Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization

World Health Organization Geneva, 1996

The International Programme on Chemical Safety (IPCS) is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

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Thallium affects all organisms, but species- and also strain-specific differences are evident. Different inorganic thallium(I) and thallium(III) compounds and organothallium compounds can show different toxicities.

The most important effect of thallium on microorganisms seems to be inhibition of nitrification by soil bacteria. Results of one study suggest that microbial community structure is disturbed at soil concentrations in the range of 1-10 mg/kg dry weight, but the form of thallium used in this experiment was not identified.

Thallium is taken up by all plant parts, but principally by the roots. After uptake into the cell, it is concentrated unevenly in the cytosol, probably bound to a peptide. Thallium concentrations found in plants depend on soil properties (especially pH, clay and organic matter content), as well as on the developmental stage and on the part of the plant. Thallium accumulates in chlorophyll-containing regions, but to a lesser degree in thallium-resistant plants. Oxygen production is reduced by thallium, presumably by direct action on electron transfer in photosystem II. Interference with the pigments is indicated by the occurrence of chlorosis. In addition, impaired uptake of trace elements seems to be involved in the mechanism of toxicity. Growth is also affected, roots reacting more sensitively than leaves or stems. These effects have been reported at concentrations as low as 1 mg thallium/kg of dry plant tissue, after exposure to monovalent forms of thallium.

Most studies of effects on aquatic organisms have used soluble monovalent thallium compounds. The lowest thallium concentration reported to affect aquatic species is 8 $\mu g/litre$, which caused a reduction in growth of aquatic plants. Invertebrates are often affected at lower concentration than fish (96-h LC_{50} values are 2.2 mg thallium/litre for daphnids and 120 mg/litre for a freshwater fish). The lowest LC_{50} value, reported after exposure for about 40 days, was 40 $\mu g/litre$ for fish.

Many cases of thallium intoxication of wildlife have been caused by its large scale application as a rodenticide. In seed-eating animals and predators the CNS and/or the gastrointestinal tract are most severely affected. These effects can also be observed in farm animals. In addition, thallium causes a loss of dorsal feathers in ducks, salivation from the nose and mouth of cattle, and reduced growth in broilers, laying hens, sheep and steers.

ATSDR Agency for Toxic Substances & Disease Registry ToxFAQs™ for Cadmium

(Cadmio (/es/toxfags/es tfacts5.html))

September 2008

CAS#: 7440-43-9

[2] (/tfacts5.pdf) PDF Version, 33 KB (/tfacts5.pdf)

This fact sheet answers the most frequently asked health questions about cadmium. For more information, you may call the ATSDR Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

Highlights

Exposure to cadmium happens mostly in the workplace where cadmium products are made. The general population is exposed from breathing cigarette smoke or eating cadmium contaminated foods. Cadmium damages the kidneys, lungs, and bones. Cadmium has been found in at least 1,014 of the 1,669 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is cadmium?

Cadmium is a natural element in the earth's crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide).

All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics.

What happens to cadmium when it enters the environment?

- Cadmium enters soil, water, and air from mining, industry, and burning coal and household wastes.
- Cadmium does not break down in the environment, but can change forms.
- Cadmium particles in air can travel long distances before falling to the ground or water.
- Some forms of cadmium dissolve in water.
- Cadmium binds strongly to soil particles.
- Fish, plants, and animals take up cadmium from the environment.

How might I be exposed to cadmium?

- Eating foods containing cadmium; low levels are found in all foods (highest levels are found in shellfish, liver, and kidney meats).
- Smoking cigarettes or breathing cigarette smoke.
- Breathing contaminated workplace air.
- Drinking contaminated water.
- Living near industrial facilities which release cadmium into the air.

How can cadmium affect my health?

Breathing high levels of cadmium can severely damage the lungs. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea.

Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones.

How likely is cadmium to cause cancer?

The Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds are known human carcinogens.

How can cadmium affect children?

The health effects in children are expected to be similar to the effects seen in adults (kidney, lung, and bone damage depending on the route of exposure).

A few studies in animals indicate that younger animals absorb more cadmium than adults. Animal studies also indicate that the young are more susceptible than adults to a loss of bone and decreased bone strength from exposure to cadmium.

We don't know if cadmium causes birth defects in people. The babies of animals exposed to high levels of cadmium during pregnancy had changes in behavior and learning ability. There is also some information from animal studies that high enough exposures to cadmium before birth can reduce body weights and affect the skeleton in the developing young.

How can families reduce the risk of exposure to cadmium?

- In the home, store substances that contain cadmium safely, and keep nickel-cadmium batteries out of reach of young children.
- Cadmium is a component of tobacco smoke. Avoid smoking in enclosed spaces like inside the home or car in order to limit exposure to children and other family members.
- If you work with cadmium, use all safety precautions to avoid carrying cadmium-containing dust home from work on your clothing, skin, hair, or tools.
- A balanced diet can reduce the amount of cadmium taken into the body from food and drink.

Is there a medical test to determine whether I've been exposed to

cadmium?

Cadmium can be measured in blood, urine, hair, or nails. Urinary cadmium has been shown to accurately reflect the amount of cadmium in the body.

The amount of cadmium in your blood shows your recent exposure to cadmium. The amount of cadmium in your urine shows both your recent and your past exposure.

Has the federal government made recommendations to protect human health?

The EPA has determined that exposure to cadmium in drinking water at concentrations of 0.04 ppm for up to 10 days is not expected to cause any adverse effects in a child.

The EPA has determined that lifetime exposure to 0.005 ppm cadmium is not expected to cause any adverse effects.

The FDA has determined that the cadmium concentration in bottled drinking water should not exceed 0.005 ppm.

The Occupational Health and Safety Administration (OSHA) has limited workers' exposure to an average of 5 ìg/m³ for an 8-hour workday, 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2008. <u>Toxicological Profile for Cadmium (/ToxProfiles/TP.asp?id=48&tid=15)</u> (*Draft for Public Comment*). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

If you have questions or concerns, please contact your community or state health or environmental quality department or:

For more information, contact:

Agency for Toxic Substances and Disease Registry Division of Toxicology and Environmental Medicine 1600 Clifton Road NE, Mailstop F-62 Atlanta, GA 30333

Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)

Fax: 1-770-488-4178

Email: cdcinfo@cdc.gov)

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

Information line and technical assistance:

Phone: 888-422-8737 FAX: (770)-488-4178

To order toxicological profiles, contact:

National Technical Information Service 5285 Port Royal Road Springfield, VA 22161

Phone: 800-553-6847 or 703-605-6000

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| | Energy of second ior | nisation | 1622 kJ.mol ⁻¹ | | | Cadmium | |
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| Lenntech BV | Discovered | | Fredrich Stromeyer in 1817 | | | Californium | |
| | | | W | | | Chlorine | |
| Rotterdamseweg 402 M 2629 HH Delft The Netherlands | Cadmium | | | | | Curium | |
| tel: +31 15 261 09 00 | | | very malleable metal. Its surface | | | Cobalt | |
| fax: +31 15 261 62 89 | many respects to zinc be | | arnishes in air. It is soluble in acids aplex compounds. | s but not in alkalis. It is similar | in | Chromium | |
| e-mail: info@lenntech.com | Applications | Cesium | | | | | |
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| | cadmium into the lung | s. Blood will transport | it through the rest of the body what from cadmium-rich food. | | | Helium | |
| | | 5.5 | who live near hazardous waste sit | tes or factories that release | | Hafnium | |
| | | nd people that work i | n the metal refinery industry. Who | | it . | Mercury | |
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| | are transported to the | kidneys. Cadmium ac | cumulates in kidneys, where it da d sugars from the body and furthe | images filtering mechanisms. | This | Hassium | |
| | | | ated in kidneys is excreted from a | | 1 | Iodine | |
| | Other health effects the | at can be caused by c | admium are: | | | Indium | |
| | - Diarrhoea, stomach p - Bone fracture | pains and severe vom | iting | | | Iridium | |
| | - Reproductive failure | and possibly even infe | ertility | | | Potassium | |

- Damage to the immune system
 Psychological disorders
 Possibly DNA damage or cancer development

Environmental effects of cadmium

Cadmium waste streams from the industries mainly end up in soils. The causes of these waste streams are for instance zinc production, phosphate ore implication and bio industrial manure. Cadmium waste streams may also enter the air through (household) waste combustion and burning of fossil fuels. Because of regulations only little cadmium now enters the water through disposal of wastewater from households or industries.

Another important source of cadmium emission is the production of artificial phosphate fertilizers. Part of the cadmium ends up in the soil after the fertilizer is applied on farmland and the rest of the cadmium ends up in surface waters when waste from fertilizer productions is dumped by production companies.

Cadmium can be transported over great distances when it is absorbed by sludge. This cadmium-rich sludge can pollute surface waters as well as soils.

Cadmium strongly adsorbs to organic matter in soils. When cadmium is present in soils it can be extremely dangerous, as the uptake through food will increase. Soils that are acidified enhance the cadmium uptake by plants. This is a potential danger to the animals that are dependent upon the plants for survival. Cadmium can accumulate in their bodies, especially when they eat multiple plants. Cows may have large amounts of cadmium in their kidneys due to this

Earthworms and other essential soil organisms are extremely susceptive to cadmium poisoning. They can die at very low concentrations and this has consequences for the soil structure. When cadmium concentrations in soils are high they can influence soil processes of microrganisms and threat the whole soil ecosystem.

In aquatic ecosystems cadmium can bio accumulate in mussels, oysters, shrimps, lobsters and fish. The susceptibility to cadmium can vary greatly between aquatic organisms. Salt-water organisms are known to be more resistant to cadmium poisoning than freshwater organisms.

Animals eating or drinking cadmium sometimes get high blood-pressures, liver disease and nerve or brain

Back to periodic table of elements chart.

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Neptunium

Oxygen Osmium

Phosphorus

Protactinium

Palladium

Promethium

Polonium

Praseodymium

Platinum

Plutonium

Radium Rubidium

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Environmental Impact and Health Effects of Cadmium

By: Dr.Badruddin Khan | Posted: Sep 13, 2008

Cadmium is a lustrous, silvery-white, ductile, very malleable metal. Its surface has a bluish tinge and the metal is soft enough to be cut with a knife, but it tarnishes in air. It is soluble in acids but not in alkalis. It is similar in many respects to <u>zinc</u> but it forms more complex compounds. About three-fourths of cadmium is used in Ni-Cd batteries, most of the remaining one-fourth is used mainly for pigments, coatings and plating, and as stabilizers for plastics. Cadmium has been used particularly to electroplate steel where a film of cadmium only 0.05 mm thick will provide complete protection against the sea. Cadmium has the ability to absorb neutrons, so it is used as a barrier to control nuclear fission.

Cadmium can mainly be found in the earth's crust. It always occurs in combination with some offensive metals and consists in the industries as their inevitable by-products. After being applied it enters the environment mainly through the ground, because it is found in manures and pesticides. Naturally a very large amount of cadmium is released into the environment. About half of this cadmium is released into rivers through weathering of rocks and some cadmium is released into air through forest fires and volcanoes. The rest of the cadmium is released through human activities, such as manufacturing. No cadmium ore is mined for the metal, because more than enough is produced as a byproduct of the smelting of zinc from its ore, sphalerite (ZnS), in which CdS is a significant impurity, making up as much as 3%. Consequently, the main mining areas are those associated with zinc.

Cadmium waste streams from the industries mainly end up in soils. The causes of these waste streams are for instance zinc production, phosphate ore implication and bio industrial manure. Cadmium waste streams may also enter the air through (household) waste combustion and burning of fossil fuels. Because of regulations only little cadmium now enters the water through disposal of <u>wastewater</u> from households or industries.

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Cadmium is strongly adsorbed to organic matter in soils. When cadmium is present in soils it can be extremely dangerous, as the uptake through food will increase. Soils that are acidified enhance the cadmium uptake by plants. This is a potential danger to the animals that are dependent upon the plants for survival. Cadmium can accumulate in their bodies, especially when they eat multiple plants. Cows may have large amounts of cadmium in their kidneys due to this. Earthworms and other essential soil organisms are extremely susceptive to cadmium poisoning. They can die at very low concentrations and this has consequences for the soil structure. When cadmium concentrations in soils are high they can influence soil processes of microorganisms and threat the whole soil ecosystem. In aquatic ecosystems cadmium can bio accumulate in mussels, oysters, shrimps, lobsters and fish. The susceptibility to cadmium can vary greatly between aquatic organisms. Salt-water organisms are known to be more resistant to cadmium poisoning than freshwater organisms. Animals eating or drinking cadmium

sometimes get high blood-pressures, liver disease and nerve or brain damage.

Human uptake of cadmium takes place mainly through food. Foodstuffs that are rich in cadmium can greatly increase the cadmium concentration in human bodies. Examples are liver, mushrooms, shellfish, mussels, cocoa powder and dried seaweed. An exposure to significantly higher cadmium levels occurs when people smoke. Tobacco smoke transports cadmium into the lungs. Blood will transport it through the rest of the body where it can increase effects by potentiating cadmium that is already present from cadmium-rich food. Other high exposures can occur with people who live near hazardous waste sites or factories that release cadmium into the air and people that work in the metal refinery industry. When people breathe in cadmium it can severely damage the lungs. This may even cause death.

Cadmium is first transported to the liver through the blood. There, it is bond to proteins to form complexes that are transported to the kidneys. Cadmium accumulates in kidneys, where it damages filtering mechanisms. This causes the excretion of essential proteins and sugars from the body and further kidney damage. It takes a very long time before cadmium that has accumulated in kidneys is excreted from a human body. Other health effects that can be caused by cadmium are: diarrhoea, stomach pains and severe vomiting, bone fracture, reproductive failure and possibly even infertility, damage to the central nervous system, damage to the immune system, psychological disorders, and Possibly DNA damage or cancer development.

About the Author

Dr.Badruddin Khan, teaches Chemistry in the University of Kashmir, Srinagar, India. He loves to enrich the most valuable raw material of the society, the students, in all possible ways. His contact details are: E. mail: khanbudr@yahoo.co.in; Phone: 00919906415909.

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JOIN ab_articles_data ON ab_articles_data.id = ab_articles_meta.id

JOIN ab_articles_stats ON ab_articles_stats.id = ab_articles_meta.id JOIN ab categories ON ab categories.cat id = ab articles meta.cat id

LEFT JOIN ab_categories as ca ON ab_categories.cat_parent_id = ca.cat_ic

WHERE `ab_articles_meta`.`id` = 560400
AND `ab articles meta`.`status_id` = '1'

0.0007 User model->get pen by article id line:1432

SELECT ab user pennames.*

FROM (ab articles meta)

JOIN ab_user_pennames ON ab_user_pennames.pen_id = ab_articles_meta.pen_
WHERE ab articles meta.id IN ('560400')

0.0008 User model->get pen by pen id line:1356

SELECT *

FROM (ab user pennames)

WHERE ab_user_pennames.pen_id IN ('60540')

0.0006 User model->get pen by article id line:1432

SELECT ab user pennames.*

FROM (ab articles meta)

JOIN ab_user_pennames ON ab_user_pennames.pen_id = ab_articles_meta.pen_ WHERE ab articles meta.id IN ('560400')

0.0007 User model->get pen by article id line:1432

SELECT ab user pennames.*

FROM (ab articles meta)

JOIN ab_user_pennames ON ab_user_pennames.pen_id = ab_articles_meta.pen_ WHERE ab articles meta.id IN ('560400') session id 3b252cbc3781bc37b160998358cdd58b

204.47.242.116 ip_address

Mozilla/5.0 (Windows NT 5.1; rv:14.0) user_agent

Gecko/201001

1343939449 last_activity